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LUMBAR DISC LESIONS

CONSERVATIVE TREATMENT*

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Ordinary conservative treatment appears to me to consist of: (1) Rest in bed, (2) corsetry, (3) a plaster jacket. Should these measures fail the question of laminectomy arises. Hospitals still exist in England where heat, massage and exercises are given as a relic of the old 'fibrositis' days, but none of these three pastimes can be dignified by the name of treatment.

My views are different. If a disc protrusion has occurred, my first stand-by is manipulative reduction; my next, reduction by traction. If that fails, epidural local anaesthesia may succeed in abolishing symptoms. If that fails, and the symptoms warrant and time is unlikely to bring relief, and there is no neurosis, laminectomy has to be considered.

After reduction by manipulation or traction, we carefully explain to each patient that cartilage, being avascular, cannot unite and that reduction of the displacement has not cured the damage to the disc itself; it has merely dealt with the displacement. He must therefore be careful to maintain his lordosis at all times, so as to avoid recurrence, and to come for immediate reduction should he nevertheless suffer pain again. Heavy workers benefit from a belt maintaining lordosis. You will notice that reduction followed by its maintenance by a corset follows normal orthopaedic principles and bears no relation to the common but illogical practice of applying the corset with the displacement still in being.

EXAMINATION

The choice of method in conservative treatment depends on the behaviour, size, situation, consistency and duration of the displacement. This is determined as accurately as possible by listening to the patient's story and by careful clinical examination. It does not rest on the X-ray appearances at all; for this shows the width of the joint space but not whether a protrusion is present or not. The past behaviour of a disc lesion is described in the history; whether it is recent or long standing, pulpy or annular, stable or not, central or posterolateral, altered or not by posture or compression, is deduced from what the patient says. The clinical examination that follows allows an estimate to be made of position and size. It is in three parts: (1) The articular signs on movement of the lumbar spine, (2) the mobility of the dura mater when stretched from above and of the nerve-roots when stretched from below, and (3) the presence or not of impaired conduction of the nerve-roots.

MANIPULATION

If a disc lesion with displacement is found present, an immediate attempt is made at manipulative reduction, unless some contra-indication exists. Were this policy followed as a matter of course, as it would be with a broken bone or the meniscus at the knee, much avoidable invalidism would, in fact, be avoided. The medical man should perform the manipulation at once himself or, as we do at St. Thomas's, delegate the work to trained physiotherapists. No anaesthesia is allowed; for this makes manipulation both dangerous and less likely to succeed. As long as the patient is conscious, the position of the protrusion can be ascertained after each manoeuvre; for straight-leg raising and pain on coughing or on lumbar movement can be tested afresh each time. In this way the manipulator can watch the protrusion moving and he is thus guided towards the most effective method; moreover these changes in the patient's clinical state show him whether to go on or to stop. Once full reduction is achieved (i.e. full range without pain) the postures to adopt are explained to the patient and the question of a support to help maintain lordosis arises.

Pringle (1956) found the period of absence from work halved when his physiotherapists adopted the methods of treating disc lesions outlined here.

* A paper presented at the South African Medical Congress, Durban, September 1957.

Contra-indications to Manipulation

1. *Danger to fourth sacral root.* Great care must be taken to avoid compression of the fourth sacral root, since any damage done may prove permanent. Manipulations should be avoided in patients with (*a*) bilateral sciatica (except in the elderly), (*b*) referred coccygodynia, (*c*) paraesthesiae in the saddle area, perineum, genitals or rectum, (*d*) interference with bladder function related to the lumbago or sciatica or (*e*) impotence.

2. *Acute lumbago.* If gentle pressure on the lumbar spine causes severe pain, manipulation is out of the question and epidural local anaesthesia should be substituted.

3. *Psychoneurosis.* Neurotic patients with a slight genuine disc lesion often assure the doctor that they are able to stand manipulation. It is carried out with care, and the patient leaves the department much better. That evening however, a nervous crisis is apt to develop, and the patient and family doctor must be warned about this.

Manipulation Harmless but Ineffective

Manipulation is useless but not harmful in the following conditions:

1. *Too large a protrusion.* Protrusions larger than the aperture whence they emerged cannot be reduced by manipulation. This is indicated by (*a*) sciatica with marked lateral deviation of the lumbar spine, or (*b*) considerable root palsy, i.e. two or more neurological signs.

2. *Too long standing.* If a protrusion has caused sciatica that has lasted longer than 6 months, manipulation is not worth trying unless the patient is over 60 years old.

3. *Nuclear protrusion.* This may be suggested by a slow onset to the symptoms, or by a history of primary postero-lateral protrusion.

4. *Signs of irreducibility.* If the trunk movements other than flexion hurt in the limb instead of the back, or side-flexion towards the painful side hurts in the back, success in manipulation is unlikely.

5. *Compression phenomenon.* If the pain comes on after the patient has stood some minutes and disappears as soon as he sits or lies, only arthrodesis avails.

6. *Post-laminectomy.* Traction is often successful in recurrence after laminectomy, but manipulation hardly ever succeeds.

TRACTION

The main indication for traction is a pulpy protrusion, or one that has unexpectedly defied manipulation. The treatment lasts ½-1 hour daily and a 40-80 kg. distracting force is used.

Contra-indications to traction

1. Lumbago with twinges. Such violent pain is often produced when the traction is abated that it takes several hours to get the patient off the couch.

2. Immediately after manipulation has failed, no benefit is likely, whereas traction carried out the next day may well initiate improvement.

3. Such respiratory or heart disease that the patient cannot bear the thoracic harness.

EPIDURAL LOCAL ANAESTHESIA

An extradural injection of 50 c.c. of 0·5% procaine is made through a lumbar puncture needle introduced *via* the sacral hiatus. It is a simple out-patient procedure, which I have carried out 20,000 times.

The indications for injection are as follows:

1. *Acute lumbago.* If manipulation proves too painful, this is the treatment of choice. Though, of course, it does not alter the displacement it destroys all pain for 90 minutes, since the protrusion now presses, *via* the posterior ligament, on a dural membrane rendered insensitive. During this time, the patient moves freely, and often initiates spontaneous reduction.

2. *Sciatica for too long.* Sciatica ought to get well of itself in a year if the patient is under 60 years old. If spontaneous recovery is delayed, 1-3 injections at about fortnightly intervals are often curative.

3. *Manipulation and traction have failed.* Clearly the presence of the displacement is unavoidable (laminectomy apart) and the best that can be done is to mitigate the pain by local desensitization.

4. *Backache.* If this is present at night or on waking only, or examination shows that the lumbar movements are only slightly uncomfortable, the injection is often curative.

5. *Referred coccygodynia.* This is required in any case for diagnostic purposes, and usually has a therapeutic effect as well.

Contra-indications to injection

1. Local sepsis.
2. Sensitivity to procaine.
3. Post-laminectomy. The channel along which the injection must flow is blocked with fibrous scar tissue.

OPERATION

Indications for laminectomy

Fourth sacral palsy (urgent).
Intractable severe pain.
Gross lumbar deformity with sciatica in a young patient.
Adherent nerve root.
Repeated crippling attacks.

Indications for arthrodesis

Compression phenomena.
Spondylolisthesis.
Repeated frequent attacks of internal derangement.

N.B. The disc must be in place at the time when the fusion is performed.

STATISTICAL SURVEY

In 1953 I saw 538 cases of lumbar disc lesions in private practice. Of these, 370 have either come to see me again in a subsequent year (8%) or have answered a postal questionnaire.

Total confirmed = 370

Causing pain in back	46·0%
Causing root pain	54·0%
Spondylolisthesis present	3·9%

PAIN IN BACK

Treated by Manipulation, 58·0%

Well in 1 session	49·0%
Well in 2 sessions	11·3%
Well in 3 sessions	6·1%
Well in 4 sessions	4·1%
Better in 1 session	12·3%
Better in 2 sessions	3·1%
Better in 3 sessions	2·0%
Same in 1 session	4·1%
Same in 2 sessions	1·0%
Same in 4 sessions	2·0%
Worse in 1 session	2·0%
Worse in 2 sessions	1·0%
Worse in 3 sessions	2·0%

*Recurred within 3 years—44·0% of those pain-free after manipulation.**

Treated by Traction, 13·0% (daily for 1-4 weeks)

Well after Traction 38·0% (12·5% in 1 week, 87·5% in 2 weeks).

Better after Traction 19·3% (50·0% in 2 weeks, 50·0% in 3 weeks).

Same after Traction 38·0% (5·0% in 1 week, 12·5% in 2 weeks, 25·0% in 3 weeks, 12·5% in 4 weeks).

Worse after Traction 4·7% (in 2 weeks—a single case).

*Recurred within 3 years—20·0% of those pain-free after course of Traction.**

Treated by Epidural Local Anaesthesia, 17·0%

Well	43·0%
Better	25·0%
Same (after initial relief)	22·0%
Worse	10·0%

*Recurred within 3 years—21·5% of those well after injection. **

SCIATICA

Treated by Manipulation, 31·0%

Well in 1 session	33·5%
Well in 2 sessions	11·5%
Well in 3 sessions	6·5%
Well in 4 sessions	6·5%
Better in 1 session	16·5%
Better in 2 sessions	3·5%
Better in 3 sessions	3·5%
Better in 4 sessions	1·5%
Same in 1 session	10·0%
Same in 2 sessions	1·5%
Same in 4 sessions	1·5%
Worse in 1 session	1·5%
Worse in 2 sessions	1·5%

*Recurred within 3 years—40·0% of those pain-free after manipulation. **

Treated by Traction, 25·0%

Well after Traction 56·0% (43·0% in 1 week, 25·0% in 2 weeks, 10·5% in 3 weeks, 21·5% in 4 weeks).

Better after Traction 24·0% (34·0% in 1 week, 16·0% in 2 weeks, 25·0% in 3 weeks, 25·0% in 4 weeks).

Same after Traction 12·0% (50·0% in 2 weeks, 33·0% in 3 weeks, 17·0% in 4 weeks).

Worse after Traction 8·0% (50·0% worse after 1 week, 50·0% worse after 3 weeks).

*Recurred within 3 years—20·0% of those well after course of Traction. **

Treated by Epidural Local Anaesthesia, 26·0%

Well	66·5%
Better	13·5%
Same	17·5%
Worse	3·5%

*Recurred within 3 years—18·0% of those well after the injection. **

Treated by Laminectomy, 3·0% (i.e. 1% of the total number of patients.

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* Includes minor recurrences which recovered without treatment.

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EDITORIAL

PHYSIOLOGICAL SURGERY

The position of the surgeon has altered considerably in the last century. One has only to recall the heroic days before anaesthesia, just 100 years ago, to understand that the surgeon of those days had to keep before him the ideal of Paracelsus, 'the eye of an eagle, the heart of a lion, the hand of a woman', so as 'not to be impressed by the cries of his patient, and not to cut less than be necessary'. In those days, when every surgical manoeuvre meant *peine forte et dure*, only the bravest heart could stand up to the terrible ordeal of operating. It is said of Cheselden, whose *métier* was cutting for stone, and who used to do this complicated perineal operation in 51 seconds, that he could not sleep the night before operating, and that he sweated profusely in the theatre before he started work. The tension on the surgeon, the patient and the onlooker was great and, since pain was maximal, the aim of the surgeon was to complete his operation as soon as possible. Liston held the incredible record of disarticulating at the hip joint in 17 seconds, and in him operating dexterity probably reached its zenith.

The age of anaesthesia brought relief, and not only to the suffering of the patient. The surgeon also now had time to think while he was operating. He became an anatomist, and not a few succumbed to the temptation to make an anatomical dissection rather than functional results their immediate goal. It required considerable judgment to tread the narrow path between making a beautiful anatomical job of every operation and performing the slap-dash hearty kind of operation which persisted from the bad old days and which somehow often gave better results than the meticulous worker achieved. Moynihan aptly referred to the three kinds of operators as 'the scratchers, the slashers and the surgeons', and liked to think of himself as an 'operating physician'. With the urgent pressure on operating time relieved, there came a tendency to undervalue surgical dexterity as mere showmanship; but even the showman with his boldness and skill still had a place in the treatment of the difficult case and in the bad anaesthetic risk. In these cases a short operating time was still vital, and the rapid surgical prestidigitator again came into his own. The more quickly he worked and the more gently he handled the tissues, the less the operating wound was exposed to bacterial contamination and the better the results. The era of the anatomical surgeon was a vital and important one in the development of the art.

VAN DIE REDAKSIE

FISIOLOGIESE CHIRURGIE

Die posisie van die chirurg het aansienlik verander in die afgelope eeu. 'n Mens hoef maar net te dink aan die heldedae voordat verdowing sy verskyning gemaak het—maar net 'n honderd jaar gelede—om te verstaan dat die chirurg van daardie dae gedurig die ideaal van Paracelsus voor oë moes hou: ‚die oog van 'n arend, die hart van 'n leeu, en die hand van 'n vrou' sodat hy ‚nie bewoë word deur die skreeue van sy pasiënt en minder as wat nodig is sny nie'. In daardie dae, toe elke snykundige operasie *peine forte et dure* beteken het, kon slegs die dapperstes die verskriklike vuurproef van opereer deurmaak. Dit word beweer dat Cheselden, wie se *métier* steenoperasies was, en wat hierdie ingewikkelde perineum-operasie in 51 sekondes afgehandel het, nooit die nag voor 'n operasie kon slaap nie, en dat die sweet hom afgetap het in die operasiesaal voordat hy begin werk het. Die chirurg, die pasiënt en die toeskouer moes die grootste spanning verduur, en omdat die pyn ontsettend was, het die chirurg hom dit ten doel gestel om die operasie so gou moontlik klaar te maak. Liston het 'n ongelooflike rekord opgestel: gewrigsafsetting by die heup in 17 sekondes. In hom het chirurgiese knaphandigheid seker die hoogtepunt bereik.

Die eeu van verdowing het verligting gebring, en nie alleen van die lyding van die pasiënt nie. Die chirurg het nou kans gekry om te dink terwyl hy opereer. Hy het 'n anatomis geword, en 'n hele paar het voor die versoeking geswig om anatomiese ontleding eerder as funksionele sukses hul onmiddellike doel te maak. Dit het aansienlik baie oordeelkundigheid geverg om die smalle paadjie te betree tussen 'n pragtige stukkie anatomiese werk aan die een kant en die halsoorkop, haastige soort operasie wat 'n oorlewering van die slegte ou dae was aan die ander kant; laasgenoemde was op die een of ander manier dikwels nog meer suksesvol as die werk van die noukeurige chirurg. Moynihan het die drie soorte snydokters raak beskryf as ‚die krappers, die kappers en die chirurge', en hy het homself graag beskryf as 'n ‚opererende internis'. Toe die chirurgie nie meer onderworpe was aan die drang van beknopte duur nie, het daar 'n neiging ontstaan om snykundige knaphandigheid te onderskat en as blote spoggery te bestempel, maar selfs die pronker met sy vrymoedigheid en vaardigheid het nog 'n waardevolle plek beklee by moeilike gevalle en gevalle waar verdowing gevaarlik was vir die pasiënt. By sulke gevalle was 'n kort operasie-tyd nog lewensbelangrik, en hier het die vinnige chirurgiese goëlaar weer bobaas gespeel. Hoe vinniger hy geopereer het en hoe sagter hy met die weefsels gewerk het, hoe korter was die snywond blootgestel aan bakteriese besmetting en hoe beter die resultate. Die tydperk van die anatomiese chirurg was 'n noodsaaklike en belangrike een in die ontwikkeling van die kuns.

Surgery, however, has taken another change in direction in the last 30 years. The conquest of infection, the control of surgical shock, and improvements in anaesthesia and in pre- and post-operative metabolic control have permitted leisurely, precise and planned operations to be performed hitherto beyond the surgeon's imagination. The operator of today, while retaining many of the characteristics of his surgical ancestor is now also an applied physiologist. His operations are based on the knowledge of physicians and physiologists, and more and more he himself is approaching Moynihan's ideal of the 'operating physician'. During this era of physiological surgery the operator has penetrated ever deeper and more widely into the organs and cavities of the body, until even the interior of the cardiac chambers, the latest bastion to fall, are now subjected to surgical enterprise.

Eiselsberg, Paget and Sauerbruch at one time or another during the last fifty years have asserted that the limits of surgical progress had been reached. Nevertheless, while anyone can see and all are aware of the great advances recently made, it remains a fact that modern surgery is a young science barely a century old; we are in reality only at the 'end of the beginning'.

Die chirurgie het egter in die laaste 30 jaar 'n ander wending geneem. Die oorwinning van besmetting, die beheer van snykundige skok, en verbeterings in verdowing en in metaboliese beheer vóór en ná die operasie het tydsame, noukeurige en beplande operasies moontlik gemaak wat voorheen die verbeelding van die snydokter te bowe gegaan het. Die moderne snydokter, hoewel hy nog baie van sy chirurgiese voorgangers se kenmerke behou, is vandag ook 'n toepassende fisioloog. Sy operasies is gegrond op die kennis van interniste en fisioloë, en hy ontwikkel al meer in die rigting van Moynihan se ‚opererende internis'. In hierdie tyd van fisiologiese chirurgie dring die snydokter al meer en al dieper in die organe en holtes van die liggaam in, en selfs die binnekant van die hartkamers, die laaste fort wat oorwin moes word, is vandag die onderwerp van chirurgiese onderneming.

Eiselsberg, Paget en Sauerbruch het almal by die een of ander geleentheid in die afgelope 50 jaar beweer dat ons die perke van vooruitgang op gebied van die snykunde bereik het. Hoewel enigeen kan sien en almal bewus is van die geweldige vooruitgang wat onlangs gemaak is, is dit nogtans 'n feit dat die moderne chirurgie 'n jong wetenskap van nouliks honderd jaar oud is. Ons is in werklikheid nog maar aan die ‚einde van die begin'.

COLLOIDAL RADIOGOLD IN MALIGNANT EFFUSIONS AND EARLY OVARIAN CARCINOMA

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Radiogold has the advantage over most other sources of ionizing radiation in that it can be used either as a solid gamma-ray emitter (radiogold seeds) or as a liquid beta-ray emitter (colloidal radiogold). Radiogold seeds can be used as substitutes for radon seeds. During the past decade colloidal radiogold has emerged as a therapeutic agent of great promise in certain malignant conditions. Owing to its short half-life, its chemical inertness and its availability in a chemically pure state, colloidal radiogold can be used intravenously, interstitially and intracavitarily.

Recurrent malignant fluid formation in the pleural or peritoneal cavities in advanced malignant disease is a troublesome problem requiring frequent paracentesis to relieve the embarrassed patient, but since the advent of colloidal radiogold a method of treatment is available that is simple and effective in controlling these recurrent effusions in the vast majority of cases. In using colloidal radiogold, however, strict precautions must be taken to protect the nursing staff and visitors from stray gamma radiation emitted by the radioactive patient.

We have used colloidal radiogold essentially as a palliative measure to control recurrent pleural and peritoneal effusions in advanced malignant disease. Recently, however, one of us (E.L.J.) has used colloidal radiogold intraperitoneally as a curative measure for early ovarian cancer and some other malignant abdominal lesions following radical surgery.

This article reviews the value of colloidal radiogold as used in intracavitary radiotherapy during the past 5 years (1953-57), (1) as a palliative measure in the treatment of 38 cases of advanced malignancy in which different primary cancers presented with recurrent troublesome malignant pleural or peritoneal effusions, (2) as a curative measure in 6 cases of early ovarian cancer following radical surgery, and (3) as a curative measure in 3 miscellaneous cases. We have treated in all 47 cases with colloidal radiogold.

REVIEW OF THE LITERATURE

Müller[1] of Zürich (1945) was the first to use suspensions of radioisotopes in the control of malignant ascites. He first used radiozinc prepared in a cyclotron, which he injected into the abdominal cavity of a patient suffering from advanced malignancy with ascites, and was able to keep the patient free of fluid for 3 years. Later[2] he used colloidal radiogold prepared in a nuclear reactor and by 1950 he was able to report encouraging results in 8 cases following the intraperitoneal administration of colloidal radiogold.

Since 1950 numerous reports have attested to the value of this radio-isotope in the control of malignant effusions. Kent *et al.*[3] in an analysis of 51 cases of malignant ascites treated with colloidal radiogold reported improvement in 60% of their cases. Walter[4] was able to control malignant ascites effectively in 7 out of 15 patients, while Storaasli *et al.*[5] found a good response in 7 out of 13 cases. Simon *et al.*[6] obtained favourable results in 7 out of 14 cases of malignant pleural effusions. Colby[7] administered colloidal

radiogold intrapleurally in 41 patients and was able to record a good result in 40%.

Kligerman and Habif[9] treated 35 cases of malignant pleural and peritoneal effusions and found that colloidal radiogold proved to be of value in 50% of those patients who had survived 1 month or more after the injection. They concluded that for the limited purpose of controlling malignant fluid formation in the serous cavities, colloidal radiogold was superior to any form of external radiation.

Dennis, Workman and Bauer[9] analyzed 58 cases of malignant serous effusions treated by them with colloidal radiogold and found that of 17 cases of malignant ascites secondary to primary malignancy of the ovary, 7 cases (41%) revealed a definite palliative benefit with an average duration of 4·5 months. Of the 10 patients who showed no improvement, all had large palpable masses when treated and 7 died within 30 days of the treatment. In 19 cases with pleural effusion secondary to proved bronchogenic carcinoma, excellent results were obtained in 15 cases (80%) with an average improvement of 5 months. Of the 8 cases of breast cancer with secondary pleural effusion, 6 (75%) showed excellent palliative results, lasting up to 11 months with an average of 5 months. In pleural effusions following lymphoma, they state that the results were surprisingly poor; only 1 case out of 5 showing any palliative response and that for a period no longer than 2 months.

Lewis[10] reported that in the treatment of ovarian cancer, he had used colloidal radiogold both as part of radical and part of palliative methods of treatment. For radical treatment he used colloidal radiogold in cases where free fluid was found in the peritoneal cavity at operation, or spill of malignant cells was suspected, or where small peritoneal seedlings were actually present. For palliative treatment he used colloidal radiogold where recurrent malignant ascites was the presenting symptom and here it was used alone or in combination with other methods. Out of 45 cases, 15 were treated from a curative point of view. Of these 15 cases, 3 have died but only one with ascites; 4 are alive and symptom-free, 29, 26, 18 and 12 months after single doses of about 150 mc. of colloidal radiogold introduced into the peritoneal cavity; 8 others are alive and symptom-free, but all these have been treated less than 1 year ago, and 4 of them less than 6 months. Lewis found that the colloidal radiogold therapy had caused only slight constitutional disturbances in these patients; some nausea without actual vomiting for one or two days seemed to be the rule, but it had not interfered materially with the subsequent general condition or drastically limited the amount of other treatment it had been possible to give them.

Elkins and Keettel[11] record their experience in the treatment of 66 cases of ovarian malignancy with colloidal radiogold from a palliative as well as a curative point of view. In 25 of these cases they had used colloidal radiogold either alone[12] or in conjunction with deep X-ray therapy[13] as a curative measure following hysterectomy and bilateral salpingo-oöpherectomy. They conclude that it was still too soon to draw any conclusions, but they were of the opinion that surgery and external radiation were inadequate for the radical treatment of ovarian cancer, particularly in early cases, and that colloidal radiogold was not only of benefit as a palliative measure in advanced ovarian cancer with recurrent malignant ascites, but that it was perhaps the best curative measure to be used in early cases, alone or in conjunction with external radiation following radical surgery.

Gwen Hilton *et al.*[12] record that during the past 3 years they have treated 100 cases of malignant effusion by intracavitary injection of colloidal radiogold and that the results could be assessed in 94 with primary growths in bronchus, ovary, breast, lymph tissue, stomach, ampulla of Vater, uterine cervix, body of uterus and colon. Of these 94 cases (*a*) 40 showed a good response (i.e. they ceased to make further effusions for a period ranging from 2 to 30 months—a few of them required more than one aspiration before the formation of fluid stopped, and in 12 cases a second injection of colloidal radiogold was required after an interval of 6 weeks to stop the fluid formation); (*b*) 8 showed a moderate response (i.e. they ceased to make effusions rapidly, so that the interval between aspirations could be extended); (*c*) 25 did not respond to treatment (in these cases, numerous or large tumour masses were present, and the authors state that they would not now consider them for treatment with colloidal radiogold); and (*d*) 21 died under 6 weeks.

Our experiences with colloidal radiogold are similar to those of other workers in this field.

THE USE OF COLLOIDAL RADIOGOLD

Source and Transport

In South Africa colloidal radiogold is obtained by ordering it through the South African Atomic Energy Board who, by law, control the distribution of radioisotopes in the Union. The colloidal radiogold arrives 7—10 days after the order has been placed. It is prepared at the Radiochemical Centre in England and is dispatched by air either as air freight or in special wing-tip compartments which require no shielding. After its arrival at Jan Smuts Airport machinery exists for transporting it to any of the major centres of the Union.

Physics

Colloidal radiogold can be prepared with high specific activity and free from radio-active contaminants. It has a half-life of 2·69 days and emits beta- and gamma-rays. The beta-rays have a maximum energy of 0·96 m.e.v., an average energy of 0·39 m.e.v., a maximum range of 4·0 mm. in tissue and a half thickness in tissue of 0·4 mm. The gamma-rays are predominantly of energy of 0·411 m.e.v.

The physiological effects are caused almost entirely by the beta-rays, which give up all their energy to the serosal surface with consequent high dosage to this surface. The gamma-rays are absorbed throughout a much larger volume, producing a more widely distributed dose of lower intensity. They can be detected externally and serve as a check on the distribution of the radiogold in the serous cavities.

By using the theory of Hine and Brownell[13] an approximate calculation may be made by making assumptions as to the distribution of colloidal radiogold in the peritoneal cavity. Assume 100 mc. of colloidal radiogold has been administered and that 80% of this becomes uniformly fixed to the serosal surfaces in the form of a thin film. The serosal surfaces are assumed to have an area of 3 square meters. The beta dosage at various depths below the surface is given in the following table:

Depth in mm.	*Dose in r.e.p.**
0·01	3,500
0·1	1,750
0·5	560
1·0	180
2·0	28

* The r.e.p. in these circumstances is approximately equal to the r.

The gamma dosage may be calculated by assuming the cavity to be spherical and to have a diameter of 16 cm., and that the colloidal radiogold has been uniformly distributed throughout this volume. For 100 mc. administered the dose in roentgens at the centre of this volume will be 1,000 r and at the periphery it will be 500 r.

Protection of Nursing Staff

According to the recommendations of the International Committee on Radiological Protection (1954) an individual may receive a maximum of 300 mr. of stray gamma radiation per week, 3,000 mr. in any consecutive 13 weeks, and 5,000 mr. per year. If only the hands and forearms are exposed, a maximum of 1,500 mr. per week is permissible.

The radiation levels around a patient who has received 250 mc. of colloidal radiogold have been calculated by assuming that the radiogold is situated in the centre of the

abdomen. Dosage rate in mr. per hour at various distances from the patient and at various times after the placing of 250 mc. of colloidal radiogold in the peritoneal cavity are given in the following table.

Day	Distance from the centre of activity		
	12 *inches*	30 *inches*	72 *inches*
	mr./h.	mr./h.	mr./h.
1st	640	100	18
2nd	490	80	14
3rd	380	60	11
4th	320	50	8
5th	230	36	6
6th	180	28	5
7th	140	22	4

Measurements made at the bedside of a patient who had received 250 mc. of colloidal radiogold were about 15% lower than those given in this table.

The time spent by nurses close to patients after surgical administration of colloidal radiogold under a general anaesthetic will depend upon the extent of the operation, the condition of the patient, and any special nursing procedures that are adopted. It would be impracticable to lay down hard and fast rules for the maximum time nurses may remain at the bedside of such patients.

The next table illustrates the order of exposure to be expected under average conditions with no shielding, after surgical administration of colloidal radiogold under a general anaesthetic. We may assume that immediately after the operation the nurse would 'special' the patient until full consciousness had been regained (about 1½ hours). This could be carried out from the head end of the bed (a distance of 30 inches from the centre of activity). Subsequently, washing, changing of dressings, bed making and feeding would take about ¾ hour during the day and about ½ hour at night, with the nurses standing about 12 inches from the centre of activity. Under these circumstances the day and night nurses would receive the following amounts of stray gamma radiation (in mr.) during the first week of nursing:

Day	Time spent near patient (*hours*)	Distance from activity (*inches*)	Gamma-ray exposure (*mr.*)	
			Day nurse	*Night nurse*
1st	1½	30	150	—
	¾	12	480	—
	½	12	—	320
2nd	¾	12	360	—
	½	12	—	240
3rd	¾	12	290	—
	½	12	—	190
4th	¾	12	240	—
	½	12	—	150
5th	¾	12	170	—
	½	12	—	120
6th	¾	12	140	—
	½	12	—	90
7th	¾	12	110	—
	½	12	—	70
Total		..	1,940 mr.	1,180 mr.

From the above table it is evident that some form of shielding is necessary to protect the nurses from the stray gamma radiation that is being emitted by the radio-active patient as a result of colloidal radiogold therapy. A lead shield ½ inch thick will reduce the radiation by about 19/20th i.e. to about 2% of the yearly maximum permissible amount.

We recommend that nurses attending closer than 3 feet to radio-active patients treated with colloidal radiogold should at all times work from behind a portable screen of ½ inch thickness of lead (Fig. 1). As the hands and forearms can

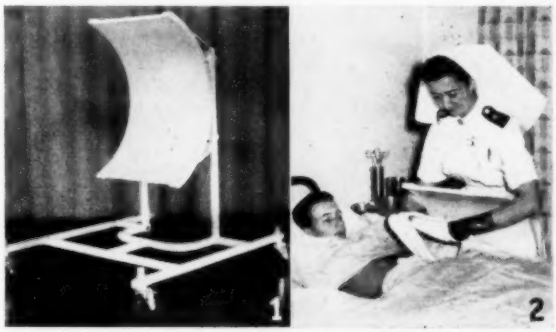


Fig. 1. Portable lead shield for nurses attending radio-active patients.

Fig. 2. Portable lead shield in use.

tolerate greater exposures of radiation, she can with safety wash and feed the patient, change the dressings, etc., provided she works from behind the lead screen, in the manner shown in Fig. 2. The portable lead screen is pushed snugly up against the bed so that the half-moon lead shield thoroughly protects the nurse attending to the patient. We also recommend that the attending nurse should wear a face mask, rubber gloves and an overall over her uniform when handling dressings that may have become contaminated with colloidal radiogold. A pocket dosimeter should be pinned to the overall and the sister in charge of the ward should keep a record of its readings, which should be consulted in assigning duties to the nurses.

Preferably all radio-active patients treated with colloidal radiogold should be nursed during the first 2 weeks in a single-bed ward, or at most a two-bed ward with maximum gamma activity per ward of 500 mc.

Young visitors should be allowed only a few minutes in the ward of a radio-active patient and during that time should not come closer than 6 feet to the patient. It would be unreasonable to restrict older visitors (past child-bearing age) in this way but, as a general rule, all visitors should abstain from sitting closer than 3 feet to the patient or staying longer than 1 hour per day during the first week.

Precautions at Post-mortem Examinations

If a patient should die soon after the administration of colloidal radiogold, the post-mortem examination should be delayed until the radio-activity had decayed to a level permitting the pathologist to work without excessive irradiation. The determination of the safe level is based upon a number of assumptions, but it should be sufficiently accurate to give the approximate order of exposure to stray gamma radiation from the radio-active body that is to be expected.

Assume: (1) Rubber gloves 0·5 mm. thick are worn, (2) time taken is 1 hour, (3) activity per unit area of the gloves is the same as that per unit area on the surface of the abdominal organs, (4) surface area of abdominal organs is 30,000 sq. cm. and (5) beta dose permitted is 750 mr.e.p. (approximately equivalent to 750 mr.).

Calculated by the method of Hine and Brownell[13] it was found that the activity in the abdomen should not exceed 10 mc. of colloidal radiogold, i.e. the post-mortem examination should not be performed until 12 days after the administration of 250 mc. of colloidal radiogold, or 9 days after the administration of 100 mc.

The radio-active body at a post-mortem examination should be placed on disposable plastic sheeting to prevent the spread of radio-active contamination, and the area should be surveyed for activity when the examination is over. All persons attending the post-mortem examination should wear rubber gloves, overall and a dosimeter pinned on the overall. A record of the readings should be kept.

The Biological Response

There is no clear explanation of how the colloidal radiogold inhibits the formation of malignant effusions. Andrews *et al.*[14] observed the disappearance of free tumour cells from the fluid after colloidal radiogold therapy in humans, and Goldie and Hahn[15] demonstrated the lethal effect of intraperitoneal colloidal radiogold on free sarcoma cells in the peritoneal fluid in mice. There now appears to be sufficient evidence to show that provided the lesions on the serosal surfaces do not exceed 2·0 mm. in depth the colloidal radiogold has a lethal effect on these small tumours and on the free cancer cells in the serous effusions. Furthermore, the inflammatory reaction produced on the serosal surfaces by the beta-rays of the colloidal radiogold mobilizes the defence mechanism of the body and many of the malignant cells and in some cases tumour masses are disposed of in this way.

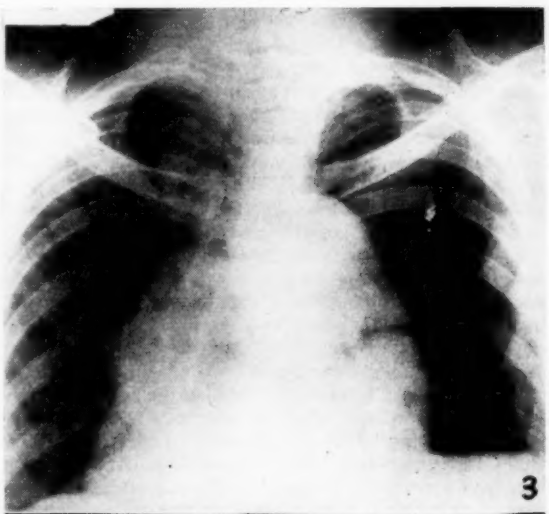
Selection of Patients

Dennis *et al.*[9] suggests as follows: 'In treating these malignant pleural and peritoneal effusions, the patients should be selected carefully for optimal results. Ideally, patients should be (1) those in whom the fluid formation has become a troublesome problem, (2) those in whom the metastases are small serosal seedlings rather than large tumour masses, and (3) those without severe constitutional effects, i.e. cachexia, anaemia, leukopenia, etc. Since the radiation effect is primarily a surface phenomenon, patients with large tumour masses are usually not effectively treated with radiogold as these large tumour masses are insufficiently irradiated. In those patients with severe constitutional effects the radiogold will often produce a rapid cessation of fluid formation but sometimes speed the downhill course.' We are in general agreement with this conception. We have, however, been surprised to see large secondary masses disappear from the pleural and peritoneal cavities after colloidal radiogold therapy administered ostensibly to inhibit recurrent fluid formation; but these cases are exceptional. (Figs. 3 and 4.)

Technique of Colloidal Radiogold Injection

Ingenious methods of protecting the staff from beta and gamma radiation during the injection of colloidal radiogold have been described by many authors, viz. Chamberlain *et al.*,[16] Andrews *et al.*,[14] Simon *et al.*,[6] Tabern *et al.*,[17] Tabern,[18] Shanks,[19] Karioris and Cowan,[20] and Gwen Hilton *et al.*,[12] to name but a few. Most departments of radiotherapy appear to have devised their own particular method of colloidal radiogold injection and protection after much trial and error. No method appears to have gained universal acceptance, except that most authorities favour the simple methods.

Lewis[10] voices the modern trend when he states: 'It is a simple matter to devise lead protection for the container; a lead bottle 2·5 cm. thick reduces the dose rate to tolerance at 7 inches (18 cm.) when holding 300 mc. Remote-control delivery devices, if they are to serve more than one case, have proved less satisfactory. The glass, however, of an ordinary Record or Luer syringe will stop all the beta radiation from the radiogold. A 20 c.c. syringe containing 150 mc. of radiogold will have a surface dose rate of about 300 mr. per minute,



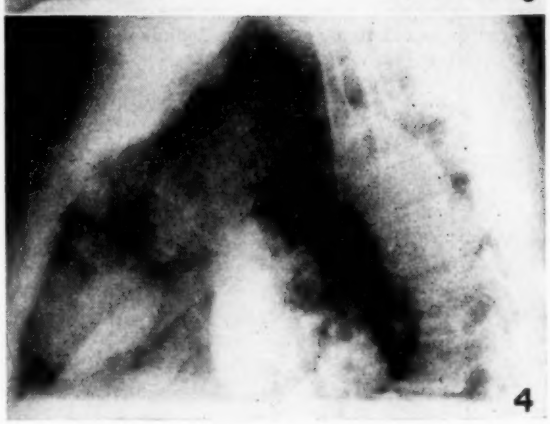


Fig. 3. Case of carcinoma of the kidney. Diagnostic pneumothorax to demonstrate, cannon-ball metastases to the pleura with malignant effusion.

Fig. 4. Same case as Fig. 3. Lateral view showing the cannon-ball metastases. After colloidal radiogold therapy these disappeared completely and the malignant effusion was inhibited. The patient is alive and well 23 months after radiogold therapy.

which would be reduced to 100 mr. per hour by a combination of 0·5 cm. lead shielding and a pistol grip making a syringe-hand-distance of 10 cm. The simplest and quickest approach to serve our purpose thus seems to be a shield protecting a standard syringe to enable a rapid and reasonably accurate injection to be made into the drip tube.'

During the past 5 years we have tried many complicated methods of injecting colloidal radiogold, but they have all been discarded in favour of the shielded-syringe method as described by Lewis.

We have found that by using a Burrell's lipiodol syringe the hands of the operator need not come closer than 5 cm. to the 3-4 c.c. of colloidal radiogold required in the syringe. The dose rate to the fingers of the operator is 160 mr. per minute while the gold is in the syringe. To reduce this to negligible amounts, namely 1·0 mr. per minute we have devised a hollow lead 'candlestick' container for the 25 c.c. Burrell's lipiodol syringe. The thickness of the lead is 0·5 cm. This special lead 'candlestick' container with syringe in position, and a second syringe next to it is shown in Fig. 5 with the bottle containing the colloidal radiogold, along with its lead shield.

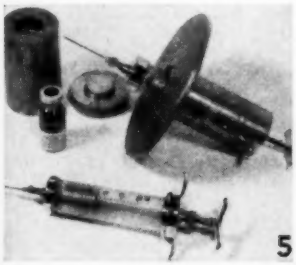


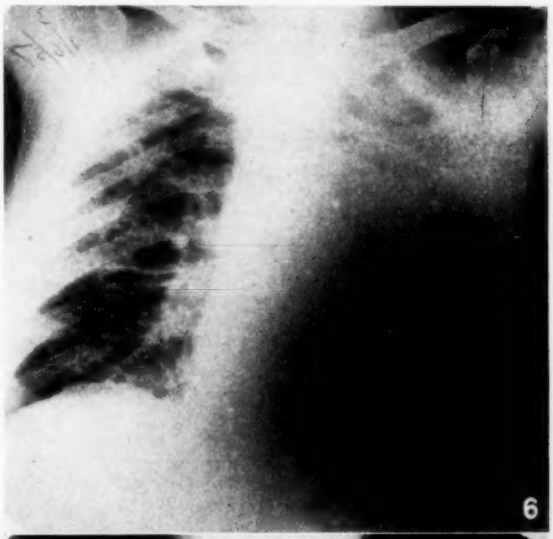
Fig. 5. Two 25 c.c. Burrell's lipiodol syringes, one placed in the lead candlestick container, with colloidal radiogold vial and lead container.

The technique that we employ for injecting colloidal radiogold into the serous cavities is as follows:

1. *For malignant pleural or peritoneal effusions as a palliative measure.* Under local anaesthesia a needle is introduced into the pleural or peritoneal cavities and as much fluid as possible is withdrawn; through the same needle, the colloidal radiogold, which is ready in a second similar syringe protected by the lead candlestick container, is introduced directly into the pleural or peritoneal cavity. The precaution is taken of withdrawing small amounts of fluid during the injection to make sure that the colloidal radiogold is reaching the pleural or peritoneal cavity and is not being injected into underlying organs or superficially into the wall of the chest or abdomen. A dose of 50-250 mc. of colloidal radiogold contained in from 3-10 c.c. of reddish-brown coloured fluid is quickly introduced and in a matter of minutes the whole operation is completed. We have found that the dose can safely be repeated in 4-6 weeks if required.

2. *For early ovarian cancer without effusion as a curative measure after radical surgery.* We follow, with some modification, the procedure described by Lewis. If at the time of the radical surgery colloidal radiogold is available, it is introduced through the same incision or preferably through a separate stab wound. If colloidal radiogold is not available at the time of operation, a small stab wound under a general or local anaesthetic is made at a later date. In the absence of ascites it is dangerous to use a puncture needle for fear of introducing the colloidal radiogold into the underlying intestines or abdominal wall. After the stab wound has been made a catheter is introduced and the peritoneum is gathered around the catheter by means of a purse string suture in readiness to close up the abdominal cavity when the catheter is withdrawn at the end of the injection. The catheter is connected to a vacolitre containing 1,000 c.c. of sterile water coloured with methelene blue. A small amount of the coloured sterile water is allowed to flow into the peritoneal cavity to make sure there is no leakage at the wound. Sterile water is preferred to normal saline, for the latter is believed to cause flocculation of the colloidal radiogold.

The colloidal radiogold is then withdrawn from the lead-protected vial and injected into the most distal part of the drip-tubing while the drip is running. Sterile water from the drip-tubing is withdrawn into the syringe at frequent interval



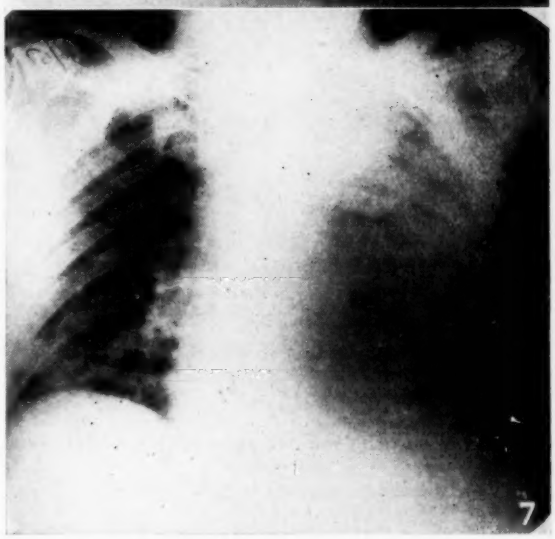


Fig. 6. Radiograph of the chest 24 hours after injection of 100 mc. of colloidal radiogold.

Fig. 7. Radiograph of the chest 120 hours after injection of 100 mc. of colloidal radiogold.

to clear the syringe completely of all the colloidal radiogold. In all, about 500-1,000 c.c. of sterile water is allowed to flow into the abdominal cavity along with 200 mc. of colloidal radiogold contained in about 5 c.c. of fluid, which is injected into the distal end of the drip-tubing in an operation time of not more than 2 minutes from the time the colloidal radiogold is withdrawn from the vial and injected into the abdominal cavity and the catheter is withdrawn and the stab wound closed.

The patient is then returned to the ward and made to lie first on one side and then on the other to obtain a good distribution of the radiogold in the abdominal cavity. The distribution can be checked, as shown by the accompanying radiographs of the chest following upon colloidal radiogold injection (Figs. 6 and 7) or by a gammagram of the abdominal cavity, which is more accurate (Fig. 8).

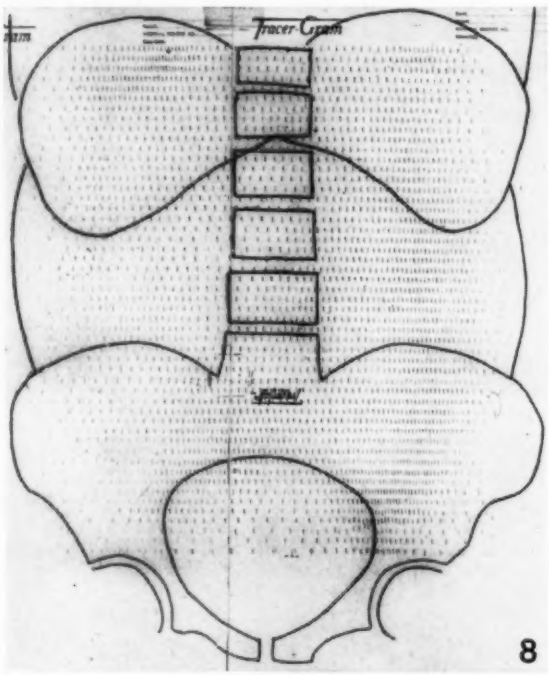


Fig. 8. Gammagram showing distribution of colloidal radiogold in the peritoneal cavity 24 hours after administration. Greater concentration on left side. There was a stab wound on the right side, and the patient preferred to lie on the left side owing to the wound pain on the right side.

Reactions and Complications

Elkins and Keettel[11] record the following reactions and complications following intraperitoneal colloidal radiogold therapy:

Nausea for a few days, sometimes associated with vomiting. A slight rise in temperature. Pain that varied from a mild discomfort relieved with aspirin to a rather severe peritoneal reaction necessitating the use of morphine. A faecal fistula occurred in one case, which they ascribed to a probable perforation of the intestine at the time of injection; it closed spontaneously without ill-effects. In one instance the entire amount of colloidal radiogold was by mischance injected into the abdominal wall. This resulted in a severe subcutaneous and skin reaction necessitating two excisions of the involved area. In 9 of their cases they had occasion to reopen the abdomen up to 14 months after the intraperitoneal injection of colloidal radiogold; in some cases they found no changes that could be attributed to the colloidal radiogold, whereas in others the peritoneum was thickened rather like that seen in chronic peritonitis. Adhesions were present with some loops of intestine adherent to other loops, but in no case did these adhesions cause obstruction. They noted that a gold discolouration of the peritoneum and outer serosal surfaces of the intestines was present in most cases. They conclude that colloidal radiogold is not an entirely innocuous agent and that intestinal changes may occur months or even years after the injection of colloidal radiogold.

On one of our cases in which a post-mortem was performed on the 10th day after the injection of a 100 mc. of colloidal radiogold into the peritoneal cavity, there was no evidence of gold discolouration of the serosal surfaces. In this case colloidal radiogold had been administered for recurrent fluid in which malignant cells had been found, but no primary lesion had been discovered. At the post-mortem there was no evidence of malignancy; the patient had died of cirrhosis of the liver. In another case, where the colloidal radiogold had been given as a curative measure after hemicolectomy for cancer of the caecum, the patient returned 21 months later and was operated on for obstruction due to adhesions. At the operation there was no evidence of gold discolouration of the serosal surfaces nor was there any evidence of metasteses. The patient made an uneventful recovery.

Excretion of the Colloidal Radiogold

If colloidal radiogold is introduced into the serous cavities, it either remains in the ascitic fluid or it is deposited on the serosal surfaces, where it decays. Only a small proportion is absorbed into the blood-stream or lymphatics. In intracavitary colloidal radiogold therapy the blood level of radioactivity remains low in contrast to the high blood level following interstitial colloidal radiogold therapy. Excretion of colloidal radiogold introduced into the serous cavities is therefore negligible and no special precautions for disposal of urine and faeces are necessary (Gwen Hilton *et al.*,[12] Andrews *et al.*[21]).

RESULTS OF COLLOIDAL RADIOGOLD THERAPY

During the past 5 years (1953-57) we have treated 47 cases of cancer either palliatively or curatively with colloidal radiogold. The colloidal radiogold was introduced into the pleural cavity in 14 cases with 5 repeats, and into the peritoneal cavity in 33 cases with 5 repeats.

Of the 47, 38 were cases of advanced malignant disease with recurrent pleural or peritoneal effusions which were treated palliatively, 6 were cases of early ovarian cancer which were treated curatively following radical surgery, and 3 do not fall into either of these categories.

1. *Advanced Malignant Disease*

In an analysis of the 38 cases the primary growth was located in the thyroid, breast, bronchus, lymph tissue, ovary or kidney, and in 5 cases the site of the primary lesion could not be established. One of the 5 died of cirrhosis of the liver with no post-mortem evidence of malignancy. Of 38, 14 presented with recurrent pleural effusion and 24 with recurrent peritoneal effusion; the results of colloidal radiogold therapy have been grouped together. A good response is recorded where the colloidal radiogold therapy produced complete inhibition of fluid formation; a fair response where fluid formation was retarded and after a second course ceased to form; and a poor response where the patient died within 2 months of receiving the colloidal radiogold therapy.

The results in the 38 cases were as follows:

(*a*) 8 (21%) died within the 2 months, some, however, in the absence of fluid.

(*b*) 16 (42%) showed a good response, some even dramatic, and in one case, the patient remained fluid-free for 31 months before death. These cases remained fluid-free for an average period of 7·5 months before death.

(*c*) 10 (26%) had recurrent effusions requiring a second course of colloidal radiogold therapy 4-6 weeks after the first course. These cases had required frequent tapping before the colloidal radiogold therapy. After the first injection fluid formation was reduced but not completely stopped, and although tapping was less frequent it still caused embarrassment. After the second injection they remained fluid-free until they died. These patients lived on an average about 4 months after the first injection and during this period fluid formation was greatly reduced and usually ceased after the second injection of colloidal radiogold. 4 (11%) are still alive, 8 months, 4 months (2) and 3 months after a single injection of colloidal radiogold, and there is no evidence of recurrent fluid formation.

These 38 patients were given from 50-100 mc. of colloidal radiogold intrapleurally or 150-250 mc. intraperitoneally. Nausea was frequently complained of after the injection, and in some cases it was followed by vomiting. The severest constitutional reactions seemed to follow intraperitoneal therapy. The reactions were never so severe as to contra-indicate colloidal radiogold therapy for advanced malignant disease.

We are of the opinion that all these advanced cases of malignant disease suffered no unjustifiable ill-effects following colloidal radiogold therapy. Although 21% must be classed as failures as far as the inhibition of fluid formation was concerned, the patients' state of health was not aggravated by the therapy. We are satisfied that 79% derived great benefit. This is most gratifying when one considers that there is no other method of treatment available (other than the less satisfactory method of using nitrogen mustard) that can bring about the inhibition of recurrent malignant fluid formation. There was never any hope of bringing about a cure in any of these cases of advanced malignant disease; nevertheless, the results have been most dramatic in a large proportion, and the patients have been able to carry on with their work right up to the very end.

2. *Early Ovarian Cancer*

The following is an analysis of the 6 proved cases of early ovarian cancer. These cases were treated with colloidal radiogold introduced intraperitoneally by one of us (E.L.J.). The colloidal radiogold was employed as a curative measure following radical surgery and in the absence of any known malignant spread. The dose administered varied from 150 to 250 mc. Each case received one dose only. They were all seen as follow-ups during the month of July 1957 and the results are tabulated as follows:

Case number	*Radiogold given*	*Dose in mc.*	*Alive and symptom-free in months after therapy*
1	9.12.53	150	43
2	10.9.54	250	34
3	24.8.55	200	23
4	6.10.55	150	21
5	29.2.56	250	17
6	26.1.57	200	6

The method of using colloidal radiogold therapy as a curative measure following radical surgery in early ovarian cancer is put on record in this article, but it is still too soon to analyse these cases any further. They are at present all in excellent health with no signs of any recurrence or other complications.

3. *The 3 cases in other categories*

(i) *Ovarian cancer with peritoneal spread.* A woman aged 45 who was diagnosed as suffering from malignant ascites was operated upon on 15 September 1954 and a primary papillary adenocarcinoma of the ovary was found, which had ruptured through the capsule and had invaded the peritoneum and omentum. There were adhesions between coils of intestine and to the peritoneal wall in the region of the splenic flexure, with a considerable amount of free fluid. A hysterectomy and bilateral salpingo-oöpherectomy was performed. The patient had a stormy convalescence after an ileus from which she recovered. On account of the presence of known residual malignancy, deep X-ray therapy was given from 12 October to 10 November. A tumour dose of 3,600r was administered in 4 weeks to the pelvic area. On 16 November 1954 150 mc. of colloidal radiogold was injected into the peritoneal cavity with the object of obtaining a good dose over the whole peritoneal cavity. This patient is alive and symptom-free 33 months later.

(ii) *Uterine cancer with peritoneal spread.* A woman aged 55 had a hysterectomy performed for carcinoma of the uterus in 1953. In 1955 she had an operation for a ventral hernia, and at operation it was discovered that she had a solitary peritoneal nodule which on microscopic examination was found to be a metastasis from the uterine cancer. On 15 August 1955 colloidal radiogold was administered intraperitoneally. She suffered a severe reaction after the gold therapy but made an uneventful recovery and is alive and symptom-free, now 24 months after colloidal radiogold therapy.

(iii) *Caecal cancer with no obvious spread.* A man aged 55 had a hemi-colectomy performed on 16 August 1955 for carcinoma of the caecum. The carcinoma had penetrated the caecum to the peritoneal covering but had not extended through the peritoneum, nor was there any evidence of peritoneal spread. In October 1955 colloidal radiogold was administered intraperitoneally. The patient made an uneventful recovery, but returned 21 months later and was operated on on 16 June 1957 for intestinal obstruction due to adhesions. There was no evidence of metastases at the operation. The patient is alive and symptom-free, now 23 months after radiogold therapy.

CONCLUSION

We are of the opinion after reviewing the literature and analysing 47 of our own cases of malignant disease treated with colloidal radiogold that this method of treatment has (1) a decided place in the treatment of troublesome recurrent malignant fluid formation in the serous cavities in advanced malignant disease, (2) that, to improve the 5 year results, it should be seriously considered as a curative measure in the treatment of early ovarian cancer following radical surgery, either, alone in the absence of any obvious malignant spread, or in conjunction with deep X-ray therapy in the presence of known residual malignancy, and (3) that it should be recommended as a curative measure in cases of possible spread in the pleural and peritoneal cavities after radical operation for cancer in these areas.

We should like to emphasize that the administration of colloidal radiogold is quite simple and safe from the patient's point of view, but that it carries a heavy hazard for the nursing and medical staffs if strict precautions are not taken in the administration, in the subsequent nursing and, if the patient dies soon after colloidal radiogold therapy, in the post-mortem examination.

We wish to express our thanks to the many medical, surgical and gynaecological colleagues who so kindly cooperated in this investigation and furnished us with details of patients; to the matron, Miss S. le Grange, and the nursing staff for their keen interest and help; to the resident engineer, Mr. L. R. Farndell and his assistants for constructing the portable lead shield; and to Mr. T. Knox for making the lead candlestick container, and finally to Mr. Theo Marais, chief photographer, for the excellent photographs.

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THYROTOXICOSIS IN AN ENDEMIC AREA*

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The problems of thyrotoxicosis concern physicians and surgeons alike. As Breitner has taught, as a result of 30 years of work on surgery of the thyroid, there is no true distinction between the surgical and medical management of this disease. It is therefore a great privilege to present to you some experiences with thyrotoxicosis which we have had as surgeons in an endemic area.

The main European goitre belt runs along with the Alps from the Rhône river to the Danube. The Tyrol, where I come from, is situated in the eastern part of the Alps. It has an area of 4,889 square miles, its highest peaks and glaciers range up to 12,000 feet. The towns in the Inn valley are 2,100 feet above sea level and some villages, well known skiing resorts, are at 6,000 feet. The water, with a hardness up to 18 degrees, is of good drinking quality but poor if not totally devoid of iodine. Especially among the hard working farmers, goitre is a serious endemic disease in spite of many years of iodinized salt supply. In Innsbruck, with 100,000 inhabitants, approximately 1,500 goitre resections are made every year out of the total population of the Tyrol of 400,000. The relative incidence of thyrotoxicosis is on an average 4%. The absolute incidence is difficult to assess. We do not agree with the traditional belief that Morbus Basedow (Graves' disease) is the disease of the non-endemic goitre area while mild hyperthyroidism is the equivalent and more frequent event in an endemic district.

When we compare our figures with those of Sallström (1935, Sweden), Thompson (1932, Chicago), Means (1948, Boston) and Fonio (1953, Switzerland) we find that thyrotoxicosis is far less modified by geographic conditions than simple goitre and there seems to be no significant difference in the variety and the severity of thyrotoxic symptoms and signs in endemic and non-endemic regions. For example there is no significant difference in the distribution of the BMR or of the pulse rate in Boston, Bern and Innsbruck and of the distribution of the classic triad and the age. The only fact distinguishing thyrotoxicosis in endemic and non-endemic areas and which is of importance for the diagnosis and the treatment is the type and the degree of thyroid hyperplasia already present at the onset of thyrotoxicosis. Whereas in non-endemic regions hyperthyroidism starts with only a slight enlargement of the thyroid gland or with no visible enlargement at all (Hunziker, 1924; Eggenberger, 1925), in the Tyrol more than 90% of thyrotaxic patients already have large nodular goitres before the onset of the disease. In other words, the specific problem of thyrotoxicosis in an endemic area is the management of toxic nodular goitres as compared to the diffuse toxic goitres which one has to treat mainly in non-endemic areas.

With rare exceptions, Tyrolean toxic goitre shows the picture of the struma diffusa et nodosa parenchymatosa with either papillary epithelial hyperplasia and lymphocytes in the stroma or no typical morphological signs of abnormal activity at all. A 'toxic adenoma' was found in only two patients (0·4%). The histological findings rarely correspond with the clinical picture. There is no doubt that thyrotoxicosis is more likely to develop in a goitrous person than in a person who has a normal thyroid gland (Riddel, 1956). In the same way a healthy thyroid gland is less sensitive towards thyrostatic treatment. The occurrence of hyperthyroidism seems to be dependent on the ability of the thyroid to compensate for a pathological stimulation. Thyrotoxicosis is reported to be unknown in places such as Bordeaux and Florence where the normal thyroid weight of 17 grams has been found (Klose, 1929).

* A paper presented at the South African Medical Congress, Durban, 1957.

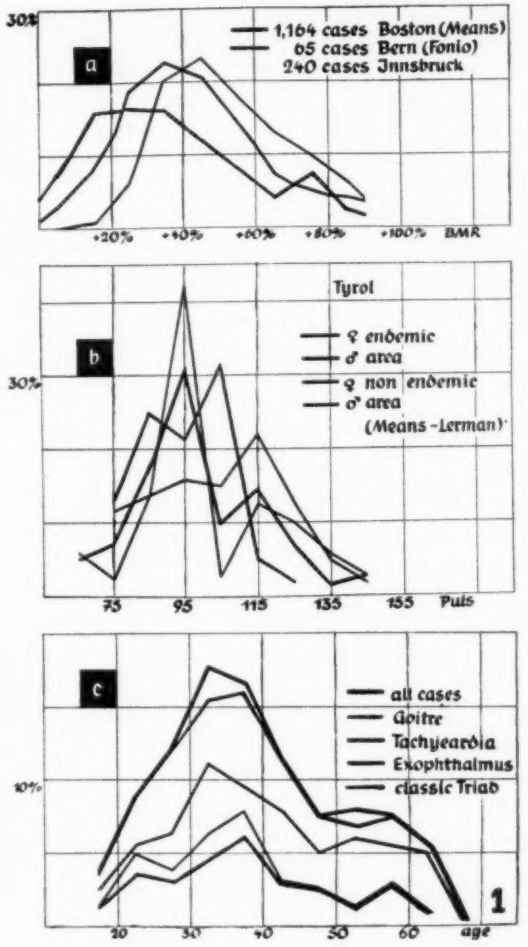

Fig. 1. Frequency distribution in 200 thyrotoxic patients of (*a*) BMR in endemic and non-endemic areas, (*b*) pulse rate in endemic and non-endemic areas, (*c*) classic Basedow signs and age in an endemic area.

DIAGNOSIS

Because of limited time at my disposal I shall restrict my discussion to the part which the predominant nodular type and the size of the goitre plays in the diagnosis and treatment of the thyrotoxicosis. In the presence of endemic goitres the correct diagnosis of thyroid function is often by no means easy. Frequently a kind of focal infection (focal toxicosis) caused by the common regressive changes, the formation of cysts or haematomas in larger nodules, a state of anxiety caused by tracheal obstruction or psycho-neurosis, neuro-circulatory asthenia, may produce a clinical picture which is perplexingly similar to many kinds of true thyrotoxicosis.

Since fatal thyroid crises have occurred after resection of even mild toxic nodular goitres, doctors become very anxious not to overlook any degree of hyperthyroidism. This leads to the practice of treating as thyrotoxic far too many nervous patients with an elevated BMR (Huber, 1950). Because of this diagnostic error euthyroid patients are deprived of their euthyroid state and thyroid enlargement is induced or—as we have observed several times—thyroid resection and thyrostatic treatment have been suggested in cases which were truly *hypo*thyroid. Furthermore, thyrotoxic patients with underlying nervous state were treated too long with thyrostatics or regarded as thiouracil- or iodine-resistant cases (Wilflingseder, 1955).

In spite of the fact that the final diagnosis has to be made on the basis of the clinical findings, a reliable thyroid function test is highly desirable in many of our patients. Especially in an endemic area it is important to bear in mind that the various tests measure different aspects of thyroid function. Thyroid function is composed of two main components, the activity of the gland and the utilization of the hormone by the cells (Breitner, 1928). The level of the serum protein-bound iodine and the many ^{131}I tests measure the activity of the gland. The cholesterol level and the BMR indicate the action of the hormone on the peripheral cells. On account of the iodine absorption, tests measuring the activity of the gland are misleading in endemic goitres in 50% of cases (Meckstroth, 1952; Rapport, 1951; Pabst and Pilz, 1956; Marcel Roche, 1957; Curtis and Fertman, 1945; Gutzeit and Parade, 1938; Holst, 1928) and are furthermore inadequate for the control of therapy when iodine or thiouracil are given (Levitt, 1954). McConahey, Owen and Keating (1956) from the Mayo Clinic tried 7 different types of ^{131}I diagnostic tests, and none of them was decisive in determining whether a patient was or was not hyperthyroid.

The cholesterol and lymphocyte test, according to personal studies on 187 persons, is in our region also of no diagnostic significance (Wilflingseder, 1952, 1954).

The BMR is easily obtained and is the largest established thyroid function test, but it is of limited usefulness in just those borderline cases of anxiety neurosis or neurocirculatory asthenia where it is most needed. The BMR technique of Robertson (1946), Fitting and Eiff (1956) is too time-consuming for clinical routine work, and has still a wide range of uncertainty. The SMR of Bartels (1948) has been found helpful by several authors (Rapport, 1951; Meckstroth, 1952; Pegni, 1949; Frases and Nordin, 1955; Leonhardt, 1953; Wilflingseder, 1948, 1955). It measures a truer basal metabolic state, because it is 'void of all nervous and muscular factors' (Bartles, 1949). Having returned from the Lahey Clinic in 1948 we studied the SMR in more than 400 cases at the Innsbruck General Hospital and have made it a routine test in border-line cases. (Wilflingseder and Villinger, 1955).

After 2 determinations of the BMR, up to 500 mg. of Narkothion* is given intravenously and 2 or 3 consecutive metabolic tests are taken while the patient is breathing quietly in a short somnolent stage. We found it not necessary to induce a deeper sleep. In the first 100 cases pentothal was used and laryngospasm occurred in 3 patients; in the last 300, with the use of Narkothion, laryngospasm was entirely avoided. Most patients wake up at the end of the second determination and are well within one hour after completion of the test. Our work at Innsbruck indicates that the range of diagnostic error using the BMR is 18-54% whereas using the SMR it is only 11-23%.

* Manufactured by Sanabo, Wien XII, Anton Scharff-Gasse.

The SMR has proved to be most helpful under the following circumstances:

1. In children, whose basal metabolic rates are notoriously unreliable.
2. In psychoneurotic patients when a basal metabolic rate determination cannot be obtained at all.
3. In all cases in which the BMR does not conform with the clinical picture and seems to be altered by extrathyroidal nervousness.
4. When hypothyroidism is suspected but masked by an elevated BMR.

The SMR is also superior to the BMR in the control of therapy (Fig. 6).

TREATMENT

The treatment of choice in toxic nodular goitre is bilateral subtotal resection. Opinions differ only as to the best pre-operative treatment. In an endemic area iodine is advised only for mild cases. We do not believe that iodine is such a dangerous weapon in the battle against goitre and thyrotoxicosis in an endemic goitre region (Breitner, 1928; Fonio, 1953) but we restrict iodine because it is effective for a quiet post-operative course in only one-third of our patients (Thompson, 1930; Means, 1948; Jackson, 1948; Wilflingseder, 1957). Our mortality remained high until the thiouracil derivatives were introduced into our country.

If the iodine-lacking case of endemic goitre receives exactly the amount of iodine lacking, say 50 or 100 micrograms, the parenchyma may become able to produce suddenly more (or a more effective) hormone, which sometimes produces a kind of hyperthyroidism known as 'Iodine-Basedow' or an exacerbation of an already existing thyrotoxicosis (Baumgartner, 1939). But if an excessive dose of iodine is given—depending on the size of the goitre, say 6-200 mg., a thyrostatic effect of some degree will result even in toxic nodular goitres. Out of 8,000 goitres only 64 cases with probable 'Iodine-Basedow' have been admitted to our department and none of these cases had received more than a small goitre-prophylaxis iodine dose.

Propyl- and methylthiouracil have had a full thyrostatic effect in all our cases treated so far. Depending on the size of the goitre, 300-600 mg. is given until a euthyroid state is reached. We have observed toxic reactions in only 3%. The mercaptan derivatives have not had a great thyrostatic effect in our nodular toxic goitres but tend to affect the haematopoietic system. In one such patient we continued treatment with the administration of 50 mg. of hexamethonium chloride 6 times a day. Within 11 days a complete remission of thyrotoxicosis was obtained. It is important to stop the thiouracil drugs for one week before operation, because leukopenia or agranulocytosis may begin several days later (Wilflingseder, 1957). We have seen a young woman die from septicopyaemia on the third post-operative day probably from this cause. We found it useful to give iodine for a week before operation not only to the diffuse toxic goitre but also to the nodular goitre. They all have a mixed parenchyma and become smaller and harder as a result of giving 50 or 100 mg. of iodine per day.

For 25 years the bilateral subtotal resection has been performed as the operation of choice in our department. Controlled hypotension was used in over 100 goitre resections (Villinger and Wilflingseder, 1955). This was helpful in certain cases but is not recommended for routine use. The thyroid remnants should not be less than 5 g. of good parenchyma on each side (30 by 20 by 15 mm.) in our endemic area. Our follow-up studies show only 2% of persistent symptoms or relapse of hyperthyroidism. They do not indicate that reduction of this figure would depend on a more radical resection. Although the ligation of all 4 arteries is preferred from the technical point of view, there was no difference in the immediate and late post-operative functional state of the thyroid when only 2 or all 4 arteries had been ligated.

In our region a relapse of thyrotoxicosis which can easily be controlled by thyrostatic drugs is regarded as a lesser evil than a permanent post-operative hyperthyroidism because we find that patients do not cooperate well on substitution therapy.

There is another technical point I should mention. Tracheomalacia because of long trachael obstruction is not infrequent in our area and tracheotomies were often necessary at the end of the operation. In a few years we have used steel ring-protheses (Wiethe, 1948) (Fig. 2) to prevent the tracheal collapse during inspiration. One or two rings are stitched to the perichondrium where the trachea has become too soft (Fig. 3). 36 patients treated in this way as reported by Haas

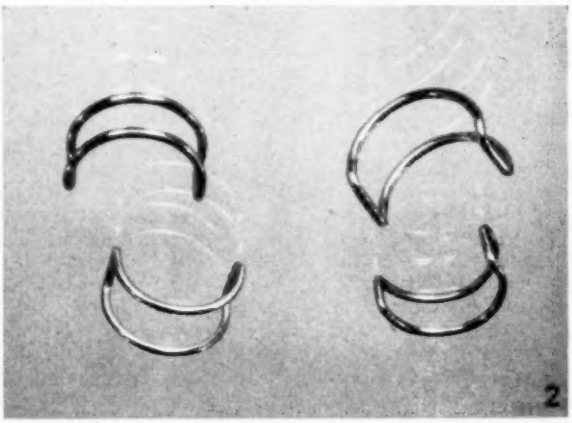

Fig. 2. Stainless steel rings used in tracheo-malacia to stabilize the tracheo post-operatively.

(1955) from our department were free from all breathing troubles and have had an uneventful post-operative course. In only 2 patients did the ring have to be removed later because of fistulas. We do not feel that tracheotomy—if done at the end of operation—is a serious complication but the steel tracheal rings safeguard respiration in cases with tracheomalacia just as well and spare the patients the burden of an additional tracheostomy after an already extensive operation.

A malignant exophthalmos developed in one patient (0·4%) 14 months after subtotal resection. Large doses of desiccated thyroid, ACTH and X-ray treatment did not stop the progress of the prostrusio bulbi. The eyes were saved by bilateral orbital decompression (Poppen, 1951).

The post-operative treatment in all thyrotoxic patients consists of simple goitre prophylaxis. Depending on the BMR and SMR, iodine or desiccated thyroid tablets are given. In purely diffuse and not too large toxic goitre, the medical regimen has been successful. Mild cases were

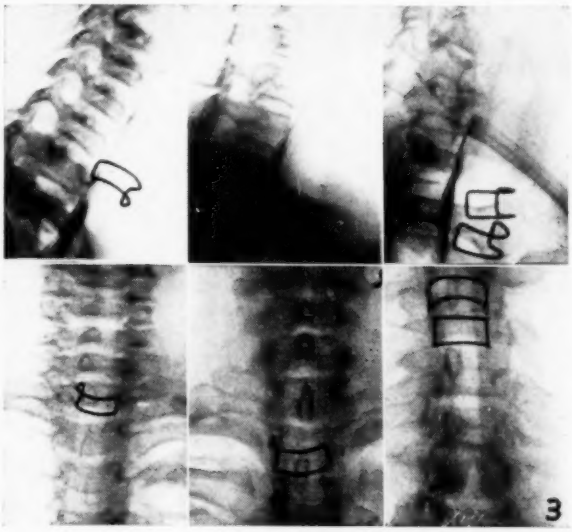

Fig. 3. Three cases showing stainless steel rings in side tube. Post-operative.

treated with desiccated thyroid only. In severe thyrotoxicosis propylthiouracil combined with desiccated thyroid is given, followed by thyroid only until the goitre has disappeared.

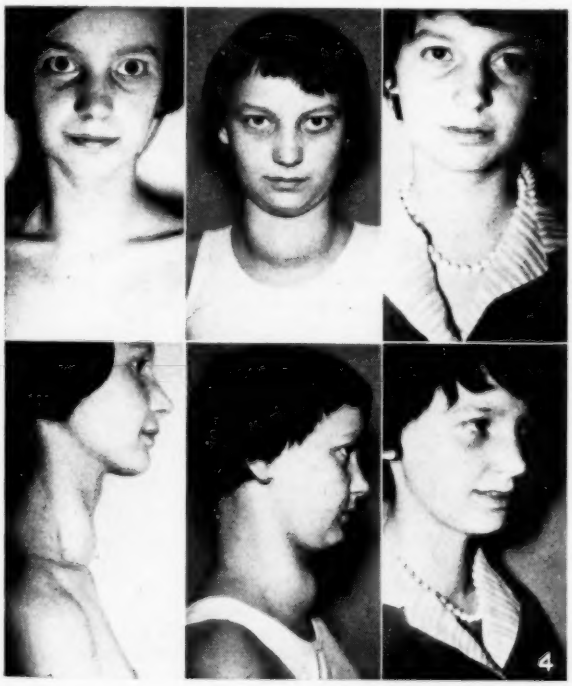

Fig. 4. A series of photographs illustrating Graves' disease in a child treated with propylthiouracil and thyreosan. The two figures on the right illustrated the end result.

CASE REPORT

This girl (Fig. 4), our only case of Graves' disease in a child in 20 years, received 200 mg. of propylthiouracil and 0·5g. of Thyreosan until euthyroidism was obtained. Thyreosan administration followed for two years. Goitre prevention is also a prophylactic against thyrotoxicosis (Eggenberger, 1926) and desiccated thyroid has been recommended by Steyrer (1917) for that reason.

SUMMARY

What is essentially the aim of surgical subtotal resection must also be attempted with our medical treatment, viz. reduction of the hyperplastic gland to a normal one. In a case of purely functional hyperplasia we may succeed with thyroid substitution therapy and cure thyrotoxicosis permanently in that way. In addition to the modern thyrostatic therapy, which has benefited our thyrotoxic patients so much, I feel we should emphasize the importance of this functional treatment especially in an endemic area.

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POLIOMYELITIS IN NAIROBI

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In November 1956 there was a sharp increase in the number of admissions of poliomyelitis to the Respiratory Unit, Nairobi. It soon became obvious that a serious epidemic was developing.

We thought that it might be of interest to readers to present an account of the racial incidence and types of cases, methods of treatment, differential diagnosis, and the causes of death. The unit accepted cases from Nairobi and from all parts of East Africa. The cases admitted from other hospitals were primarily life-threatening. Many were transported by air, a flying team being sent out from the unit for this purpose.

The unit possesses 4 Radcliffe and one Engström intermittent positive-pressure respiration (IPPR) pumps, 4 tanks, one Spiroshell cuirass respirator and 2 rocking beds. During the epidemic one Beaver IPPR pump and 7 additional tanks were borrowed. At the height of the epidemic 5 IPPR machines, 6 tanks and 2 oxygen tents were in use at the same time.

Cases Admitted

224 cases were admitted to the unit during the period November 1956 to September 1957. Only one of these cases was non-paralytic. Of this total 52 were Europeans, 18 Asians and 154 Africans.

Age Groups

The age and sex incidence of each racial group are presented in Tables I and II. As can be seen there was a considerable difference between the age distribution in the different races. In the African group, 146 of the 154 admissions (94·8%) were under the age of 5 years; of these 146 cases 115 were under 2 years of age. In the European group 28 of the 52 admissions (53·8%) were over 20 years of age. All but one of the 18 Asians admitted were under 15 years of age.

Types of Cases

Among the 154 Africans, 39 could be classified as life-threatening; the respective figures for Europeans and Asians were 19 out of 52 and 6 out of 18.

For the purpose of this paper we have used the following definitions of life-threatening poliomyelitis:

'Bulbar paralysis': pharyngeal paralysis with inability to swallow.

'Bulbo-spinal paralysis': pharyngeal paralysis plus involvement of the muscles of respiration.

'Spinal paralysis': paralysis of intercostals and/or diaphragm.

OUTSTANDING FEATURES

African Cases

Half of the Africans had severe diarrhoea on admission and some were considerably dehydrated. Many were anaemic; one had a haemoglobin level of 35% (Sahli) in spite of profound dehydration.

A distinctive feature among the African cases which was not seen in the other groups was the high proportion of 'wet lungs' (20 out of the 39 life-threatening cases). This condition was mainly due to paralysis of the abdominal muscles with or without diaphragmatic or intercostal paralysis, leading to an inability to cough. Measles or an upper-respiratory-tract infection superimposed upon this condition, or nursing in the supine position, soon led to a state of 'wet lung' with generalized rales and, in some cases, consolidation and/or atelectasis. Some of these cases gave a history of a febrile illness with paralysis of the limbs some weeks before and had been admitted to a general hospital as cases of bronchitis or bronchopneumonia before being sent to us.

Asian Cases

Of the 6 'life-threatening' cases, 2 showed an encephalitis as the predominant feature; one other had bulbo-spinal poliomyelitis and the remaining 3 had involvement of the muscles of respiration. An Asian youth of 15 years who showed a bulbar paralysis with polio-encephalitis appeared to have paralysis of the respiratory centre. He was unable to swallow and had had a tracheotomy performed before admission to the unit. During transit over a distance of 100 miles he had stopped breathing and had needed artificial respiration. On admission he began to breathe on his own. This spontaneous respiration lasted for some 20 hours, during which time he maintained a normal oxygenation of arterial blood. There was no evidence of paralysis of his intercostals, diaphragm or arms and legs. With no warning of respiratory failure he again stopped breathing and required IPPR treatment. Arterial blood analysis taken within a few minutes of this acute respiratory failure showed a pH 7·31 and Pa^*CO_2 60 mm. Hg, but the respiratory centre did not appear to respond to this degree of respiratory acidosis.

* For this symbol for arterial CO_2 tension see *Federal Proceedings* (1950): **9,** 602.

It might be argued that this respiratory acidosis was primarily due to paralysis of respiratory muscles but the respiratory failure was of extremely abrupt onset and there was no evidence of respiratory obstruction.

European Cases

In this group there was a high incidence of bulbar and bulbo-spinal involvement (12 out of the 19 life-threatening cases).

The total admissions included 14 married European women, of whom 6 were pregnant. One miscarried at 3 months, one had a stillbirth at 36 weeks and 3 live babies were delivered at term, one by Caesarean section. The remaining patient is 6 months pregnant and has been an in-patient for 3 months. The Caesarean baby was delivered during the period of infectivity and the virus was isolated from the meconium and cord blood. The virus isolations were done by Dr. J. Gear in Johannesburg and both proved to be Type I.

Of the 10 adult European males admitted, 8 showed respiratory involvement, 6 requiring respiratory aids.

TREATMENT

Statistical details of treatment are shown in Table III (A, B, C and D). Further particulars are given below:

1. *Bulbar Paralysis*

These cases were treated by posture (i.e. in the face-down position and by raising the foot of the bed), pharyngeal suction and tube or parenteral feeding. Seven cases were so treated with no deaths.

2. *Bulbo-spinal Paralysis*

Seven severe adult cases were treated with tracheotomy and IPPR machines in the manner described previously by many writers.[6, 10, 9, 4] Of these; 5 were first nursed in tank respirators until bulbar paralysis developed; tracheotomy was then performed and the patient ventilated by IPPR. Two severe cases in children both under the age of 2 years were treated in the face-down position without tracheotomy, in a tank specifically designed to allow the use of this position. There were 2 deaths in this group of 9, both being adults with severe bulbo-spinal encephalitis.

Two less severe cases in children under the age of 5 were successfully treated with posture, tube feeding and suction, and were nursed in oxygen tents. One adult female was successfully treated with posture, tube feeding and suction.

3. *Spinal paralysis, i.e. paralysis of intercostals and/or diaphragm*

Of the 17 cases who needed respiratory aids, 16 were treated in tanks and one by IPPR as he was admitted with total atelectasis of one lung which it was not possible to clear in a tank. Adult cases were put into tank respirators when their vital capacities fell to 30% of the expected normal. Of the 16 treated in tanks, 5 died (all infants under 2 years of age). The moment to use tank respirators in infants was judged by a falling or increasing respiratory rate and pulse rate, and any other indications of respiratory distress such as the use of accessory muscles and alae nasi.

Of the cases who did not need respiratory aids, 12 were treated with posture and suction and nursed in oxygen tents, and 11 others were treated by posture and suction alone.

Four infants whose predominant paralysis was of the abdominal muscles were treated by abdominal binders, posture and oxygen tents.

All cases with 'wet lungs' were given antibiotics.

Hypothermia

Seven cases were treated by hypothermia of varying degree. This form of treatment was only used in cases showing hyperpyrexia (temperature of over 104°) where previous experience has shown that a high mortality rate could be expected.[2] Lassen[7] found a mortality rate of over 90% in these cases.

There were 2 deaths in the 7 cases so treated.

Cross Infection

Considerable inconvenience was occasioned by cross infection. A *Staphylococcus aureus* phage type 80 was isolated among other bacteria from tracheotomy wounds, bronchial secretions, and infected urines. Four members of the staff developed acute septic conditions of the hands; 3 were of a serious nature. Again a *S. aureus* phage type 80 (penicillin resistant) was isolated from each case.

DIFFERENTIAL DIAGNOSIS, ETC.

Among the cases admitted as suspected poliomyelitis were the following:

Landry's paralysis in 2 cases showing bulbar spinal paralysis; these were successfully treated with cortisone; one case required respiratory aid. *Herpes zoster* in a middle-aged European with an abnormal CSF; the predominant features were severe pain in one leg with paralysis followed by the typical herpetic eruption. Three cases of *benign lymphocytic chorio-meningitis*. A case of *post-mumps encephalitis* in a European adult. Two cases of *pertussis encephalitis* in African infants. Two *hemiplegics*, both being young African males. Two African infants showing *osteomyelitis of the femur*. A European adult female with a *Coxsackie infection*, who showed severe pain of the intercostals and diaphragm which embarrassed respiration. A fatal case of *pneumococcal meningitis* with a normal CSF in a European schoolboy. A European adult with *cerebral malaria*. An African adult with *transverse myelitis*. Two European adult females with *hysteria*.

C.S.F. Findings

Some of the CSF findings did not fit into the so-called classical picture. The cell count ranged from 5 to over 1,000. In cases showing these extremes of the cell count poliomyelitis was not diagnosed unless typical paralysis was evident.

Causes of Death

Out of the total of 223 paralytic cases there were 64 with life-threatening disease. This high incidence was occasioned by the selective nature of our admissions; the great majority of cases from hospitals outside of Nairobi were of a serious nature. Of the 64, 11 died, 8 deaths occurring in the African group. Of these 7 showed 'wet lungs' on admission.

Post-mortem examinations were performed in 10 cases, in 7 of which there was obstruction to respiration in the lower respiratory tract due to muco-pus in 6 cases and to inhaled milk in the 7th. In one other case the predominant

TABLE I. TOTAL ADMISSIONS* BY RACE, SEX AND AGE

Age	African			Asian			European		
	M.	F.	Total	M.	F.	Total	M.	F.	Total
0– 1	32	23	55	1	0	1	1	0	1
1– 2	37	23	60	4	1	5	3	0	3
2– 5	17	14	31	2	5	7	2	4	6
5–15	2	2	4	3	1	4	8	1	9
15–20	1	1	2	0	1	1	3	2	5
20–30	0	2	2	0	0	0	3	14	17
30–50	0	0	0	0	0	0	7	4	11
Total	89	65	154	10	8	18	27	25	52

* 11 months ending September 1957

TABLE II. LIFE-THREATENING CASES AND DEATHS, ETC. BY RACE AND AGE

	0–5 years			5–20 years			20–50 years		
	Af.	As.	Eur.	Af.	As.	Eur.	Af.	As.	Eur.
Total	146	13	10	6	5	14	2	0	28
Resp. cases *	36	4	1	3	2	1	0		17
Resp. cases as % of total	24·6%	30·7%	10%	50%	40%	7·7%	0		60·7%
Deaths	7	1	0	1	0	0	0		2
Mortality rate	19·5%	25%	0	33·3%	0	0	0		11·7%
Age group as % of all ages	94·8%	72·2%	19·2%	4%	27·8%	27%	1·2%		53·8%

* Life-threatening poliomyelitis

TABLE III A. AFRICAN CASES AND DEATHS BY CLASSIFICATION AND TREATMENT AND BY AGE

Age (years)	B.	B.S.	Abd.	Sp.	Total	Posture etc.	Posture +O_2	Tank	Trache-otomy	Deaths
0– 1	2	0	3	10	15 (3)	5	5 (1)	5 (2)	0	3
1– 2	1	1	1	12	15 (4)	3	5	6 (3)	1 (1)	4
2– 5	0	2	0	4	6	2	2	2	0	0
5–15	0	0	0	3	3 (1)	0	2 (1)	1	0	1

TABLE III B. EUROPEAN CASES AND DEATHS BY CLASSIFICATION AND TREATMENT AND BY AGE

Age (years)	B.	B.S.	Sp.	Total	Posture etc.	Tank	IPPR	Deaths
1– 2	0	1	0	1	0	1	0	0
5–15	1	0	0	1	1	0	0	0
20–30	1	5	5	11 (2)	4	2	5 (2)	2
30–50	2	2	2	6	3	1	2	0

TABLE III C. ASIAN CASES AND DEATHS BY CLASSIFICATION AND TREATMENT AND BY AGE

Age (years)	P.E.	B.S.	Sp.	Total	Posture etc.	IPPR	Deaths
0– 1	0	0	1	1	1	0	0
1– 2	0	0	1	1	1	0	0
2– 5	1	0	1	2	2 (1)	0	1
5–15	1	1	0	2	1	1	0

TABLE III D. ALL CASES AND DEATHS BY CLASSIFICATION AND TREATMENT AND BY AGE

Age (years)	P.E.	B.	B.S.	Abd.	Sp.	Total	Posture etc.	Posture +O_2	Tank	IPPR	Trache-otomy	Deaths
0– 5	1 (1)	3	4	4	29 (7)	41	13 (1)	12 (1)	14 (5)	0	1 (1)	8
5–15	1	1	1	0	3 (1)	6	2	2 (1)	1	1	0	1
20–50	0	3	7 (2)	0	7	17	7	0	3	7 (2)	0	2
Total	2 (1)	7	12 (2)	4	39 (8)	64	22 (1)	14 (2)	18 (5)	8 (2)	1 (1)	11

P.E. = Polioencephalitis. *B.* = Bulbar paralysis. *B.S.* = Bulbo-spinal paralysis. *Abd.* = Paralysis of abdominal muscles, without intercostals or diaphragm. *Sp.* = Paralysis of intercostals and/or diaphragm with or without abdominal muscles. *Posture etc.* = Prone position, elevation of foot of bed, pharyngeal suction. *Posture* +O_2 = Above plus oxygen tents. Deaths in brackets.

findings were in the brain, no other obvious cause of death being found. One case, who died within 24 hours of the cessation of induced 'mild hypothermia',[1] showed a massive thrombosis in the right auricle extending through the tricuspid valve into the right ventricle and from there into the pulmonary artery.

Another case died during induced 'moderate hypothermia'[1] and showed changes not dissimilar to those described in experimental animals by Knocker (1955) and Grey (1957).[3]

Of the 8 deaths in African children, 4 occurred while the children were in tanks and 4 in cases treated by posture, suction, antibiotics, and oxygen tents. One of these latter cases had a tracheotomy.

Respiratory Obstruction by Secretions

The most serious difficulty in the treatment of life-threatening poliomyelitis in African infants during this epidemic was not the poliomyelitis *per se* but the lower-respiratory-tract obstruction caused by the accumulation of secretions. In 3 infants who died the main respiratory muscles involved were the abdominals, whilst involvement of the intercostal muscles or diaphragm was minimal. In many of these cases it was difficult to decide whether or not a respiratory aid was necessary. All had raised respiratory rates and some were slightly cyanosed. Tracheotomy was performed on one of these infants to facilitate suction; this was one of the 3 infants who died.

In these cases of 'wet lung' where evidence of respiratory paralysis was insufficient to warrant the use of a respiratory aid, treatment by posture, abdominal binders to help coughing and nursing in an oxygen tent seemed reasonably effective.

Improvement in Mortality Rate

In the past 5 years the mortality rate for life-threatening poliomyelitis has declined. Among the factors responsible for this can be listed the introduction of IPPR by Bjorn Ibsen[5] in 1954.

It has been suggested by Trueta[11] that tracheotomy should be avoided if possible in acute poliomyelitis, not because the mortality rate is increased, but because weaning is made more difficult. In our experience tracheotomy with IPPR in adults is sometimes a life-saving measure, though this method carries with it its own specific complications and difficulties and requires considerable experience to achieve success. This is possibly shown by figures from our own unit: In our first season we treated 5 adults with IPPR with 3 deaths (60% mortality), whereas this season 8 adults were so treated with 2 deaths (25% mortality). Other factors that may have contributed have been more detailed investigation of electrolyte and fluid balances and an improvement in hypothermic techniques.

In our first season 3 patients showed hyperpyrexia, and all 3 died. This season 7 patients showed sustained temperatures of over 104°F; they were all treated with hypothermia and 2 of them died.

Hypertension in acute poliomyelitis may be alarming, e.g. a systolic pressure of over 220 mm. Hg. In many cases this would appear to be central in origin, or at least not due to hypercarbia, for in 3 of our cases who showed high blood pressure readings the arterial CO_2 levels were normal.

Lassen[8] states that the mortality rate is increased in cases showing hypertension. Our 3 cases were treated with subcutaneous Ansolysen, which effectively reduced the blood pressure. All cases survived.

SUMMARY

A brief description of some of the features of the 1956-57 epidemic of poliomyelitis in Nairobi are presented, particular attention being paid to the age incidence in the various racial groups.

The importance of impairment of coughing due to paralysis of the abdominal muscles is emphasized as a cause of respiratory embarrassment.

The methods of treatment used in the Respiratory Unit, Nairobi, are summarized and a brief analysis of the causes of death is given.

Some reasons for the improvement in present-day mortality rates are put forward. Among these are thought to be the introduction of intermittent positive-pressure respiration, induced hypothermia, detailed attention to electrolyte and water balances, and the use of hypotensive drugs.

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OBJECTIVE PSYCHOTHERAPY: SOME THEORETICAL CONSIDERATIONS

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Neurotic behaviour has been defined as 'persistent unadaptive learned behaviour in which anxiety is almost always prominent and which is acquired in anxiety-generating situations.'[1] Behaviour which is learned can also be 'un-learned'. The processes by which responses are ordinarily diminished in magnitude and frequency of occurrence are 'extinction' and 'inhibition'. Extinction of a response is gradual and occurs after many unrewarded evocations. Non-adaptive responses which do not disappear in time by the process of extinction are, by definition, neurotic. A distinguishing feature of neurotic behaviour is in fact, its resistance to the normal process of extinction.[2]

INHIBITION OF NEUROTIC BEHAVIOUR

Neurotic behaviour is, however, open to modification and elimination by the process of inhibition, which may be defined as the imposition

of a new response (or response tendency) upon an older one such that the old one is no longer evoked. The numerous types of psychological inhibition which have been observed or postulated include proactive, retroactive, external, reciprocal, reactive and conditional inhibition. For several reasons, mainly of a practical nature, conditioned inhibition has received the greatest amount of attention in psychotherapy. Conditioned inhibition is generated when stimuli are associated with the cessation of a response in the presence of reactive inhibition (a negative drive tending to cause cessation of activity).[3] Conditioned inhibition is acquired in the same way as positive behaviour patterns are learnt. It increases progressively as a function of the number of rewarded or reinforced trials and like all habit patterns is relatively permanent. It does not dissipate spontaneously even over long periods of time. Because of these characteristics, conditioned inhibition has been widely employed by psychotherapists in their attempts to eradicate neurotic behaviour.

Wolpe's technique of psychotherapy is an attempt to produce a conditioned inhibition of neurotic behaviour by the repeated simultaneous presentation of incompatible response tendencies (reciprocal inhibition). This usually takes the form of presenting anxiety-producing stimuli while the patient is deeply relaxed in the consulting room. In this way, the tendency to respond anxiously to the noxious stimulus (e.g. blood) is superseded by the stronger and incompatible relaxation response. Repeated doses of this reciprocal inhibition (which is by itself temporary in effect) in the consulting room will steadily build up a *permanent* conditioned inhibition of the neurotic behaviour. This type of learning process has been demonstrated experimentally by various workers.[4, 5] Clinically, it has been employed by Jones,[6] Max,[7] Wolpe,[1] and Lazarus and Rachman.[8]

For every behaviour pattern there is another type of behaviour which is incompatible with the first. The task of therapy is to find an acceptable response pattern which is antagonistic to the neurotic activity of the patient and to substitute this adaptive behaviour for the non-adaptive, neurotic behaviour. Wolpe has proposed relaxation or feeding or avoidance or sexual or assertive responses as possible substitutes for neurotic behaviour, according to the requirements of the case.

ANALYTIC AND NON-ANALYTIC THERAPIES

An objection which is frequently presented by psychoanalytic-orientated critics of neo-behaviourist therapy is the concept of 'basic causes'. They argue that non-adaptive therapies deal only with symptoms and leave the basic cause or causes of the neurosis untouched; that this 'superficial approach' to the treatment of neurotic behaviour is destined to bring about only temporary alleviation of symptoms (at best) and may well aggravate the patient's condition. They claim that it is only when the 'inner forces of the psyche' have been restored to harmony by free association, transference and interpretation that the person is normal again. The major objections of the analysts may be summarized as follows: Neo-behaviourist therapy (*a*) is superficial, (*b*) is symptom-oriented, (*c*) ignores the deep inner causes of the neuroses, (*d*) can effect only temporary improvements, and (*e*) smothers certain symptoms only to provoke other new ones.

Neo-behaviourist therapy is not superficial if by this is implied either that such treatment is 'not complete' or that it can be applied with success only in certain *minor* types of behaviour disorders. There is considerable clinical and experimental evidence which proves, on the contrary, that such therapy is both complete and capable of being applied in many types of disorder, including those which analysts would regard as 'deep-seated', e.g. phobic states and anxiety neuroses of long-standing. Examples of therapeutic successes with enuretics,[9] hysterics,[10] stutterers,[1, 8] drug-addicts,[11] homosexuals,[7, 11] phobic states,[6, 1, 10, 8] alcoholics,[12] and tension-states,[13] have been reported in which the 'superficial approach' has provided *complete* or near-complete recovery. In many of the cases referred to here, the improvement has been obtained without either therapist or patient knowing what the 'basic cause' of the illness was. A particularly striking example of such a case is provided by Wolpe:[10]

'A 37-year-old miner was seen in a state of intense anxiety. He had a very marked tremor and a total amnesia for the previous 4 days. He gave a story that his wife, on whom he was greatly dependent, had cunningly got him to agree to "temporary divorce" 6 months before, and was now going to marry a friend of his. No attempt at this juncture to recall the lost memories. The patient was made to realize how ineffectual his previous attitudes had been and how he had been deceived. As a result he angrily "had it out" with his wife and a few others, anxiety rapidly decreased, and he soon felt strongly motivated to organize his whole life differently. At his 5th interview (10 days after treatment began), he said that he felt "a hundred per cent" and looked it, and was full of plans for the future. *Yet he had still recalled nothing whatever of the forgotten 4 days*'. The patient later recalled the lost memories under hypnosis. 'No important consequences ensued. A few months later he married another woman and was apparently very well adjusted generally.'

Other examples are provided by Lazarus and Rachman,[8] Mowrer,[9] and Salter.[11]

Can this evidence be taken to mean that a knowledge of the causative factors is unnecessary? The answer to this problem would appear to be a qualified affirmative. In some instances it seems unlikely that improvement in the patient's condition can be effected without such knowledge. On the other hand it would appear from the numerous therapeutic failures reported by analysts and other therapists that in certain cases insight and interpretation do *not* assist. A very obvious example of such a state of affairs can be observed in the treatment of psychopathy. An appraisal of the data leads us to the conclusion that while a knowledge of the causative process and genesis of the individual neurosis can be of considerable value in therapy, improvement can nevertheless be obtained in many cases without such knowledge.

Too great a concern with 'underlying causes' may under certain circumstances even impede therapeutic progress. The case of the miner treated by Wolpe and quoted above is one such instance. The 'forward-looking approach' as opposed to the historical technique of psychoanalysis has much to recommend it. It is quite conceivable that a patient with some pressing, immediate problem (e.g. pending divorce) may receive a severe and unnecessary jolt from the apparent lack of concern of the non-directive therapist. Several instances of intense frustration and annoyance reported by patients who have been to non-directive therapists only to have their difficulties apparently ignored or brushed aside have been observed by Lazarus.[14]

With regard to the observation that objective psychotherapy is symptom-oriented, this is generally true. The treatment of the symptom or symptoms is quite logically one of the first considerations of the psychotherapist. In numerous cases there is little else that is required as 'the deep inner causes', if they exist, cease to be relevant. The 5 cases reported by Lazarus and Rachman all bear this contention out. In case 3 the precise reason or reasons for the ambulance-phobia developing in this 13-year-old boy were never discovered. The fear was inhibited and extinguished by systematic desensitization and this *removal of the symptom* was sufficient. Case 4 was treated successfully for his stutter and again no 'deep inner causes' were revealed.

Does objective, non-analytic psychotherapy effect only temporary improvement? There is some evidence that improvements obtained by these techniques are long-lasting or permanent, but it must be admitted that the design of research work in the field of therapy, *both objective and psychoanalytic*, has been for the most part, inadequate in this respect. There is not sufficient evidence either way to justify a categorical answer. The available data on this point is provided by Wolpe,[1] Rachman and Lazarus[8], Mowrer,[9] Salter,[11] and others. Much of the evidence presented to date however, is of an anecdotal and uncontrolled nature.

Objective psychotherapy has also been criticized on the grounds that it merely smothers the neurotic symptoms. Because the 'basic causes' of the maladaptive behaviour have not been treated, it is said that new symptoms will necessarily arise to replace the extinguished behaviour patterns. For example, training an enuretic to relieve himself in the lavatory or teaching a stutterer to speak fluently will merely result in the patient 'adopting' some new deviant response. As there is no detailed evidence regarding the nature and frequency of this phenomenon it is difficult to assess its importance. While such 'transfer' of symptoms does undoubtedly occur, its frequency has probably been unduly exaggerated. In those cases where transferred symptoms arise the therapeutic procedure is quite uncomplicated. The therapist after having de-sensitized the patient to the first noxious stimulus situations when confronted with a so-called 'replacement-symptom' proceed,

to de-sensitize this new symptom in turn. When this treatment has been successfully completed, the probability of recurrence is extremely slight. It will be agreed that all neurotic symptoms in the patient have some degree of interdependence and the weakening or extinction of any one symptom is likely to affect all the others in like manner. The symptom which is treated first is usually the most resistant.* Behaviour patterns treated subsequently are more easily modified. If a new symptom arises it can be expected to be of rather weaker strength and hence readily amenable to inhibition or extinction. This 'symptom-replacement' phenomenon and its treatment has been described by Lazarus and Rachman.[6] Their case 5, a married woman of 29, had developed a phobic reaction to dogs as the result of a traumatic incident 5 years earlier. After 3 years of psychoanalysis her fear of dogs had disappeared but instead she had developed a chronic anxiety state with numerous, varied phobias ('symptom-replacement'). After 6 weeks of intensive psychotherapy (28 sessions) she was much improved, but her dog-phobia returned. After a further 28 sessions devoted mainly to the inhibition of this phobia, she was discharged 'much improved'. A year later she was still healthy and the extinction of the dog-phobia had been maintained. This case-history illustrates the treatment of 'symptom-replacement' by objective psychotherapy and also the development of 'replacement' with psychoanalysis.

* Case 1 reported by Lazarus and Rachman[6] required 21 sessions before the elimination of the first anxiety hierarchy and only 4 for the last in the series.

It is contended that objective psychotherapy is of considerable promise and that none of the criticisms presented to date are damaging. Objective psychotherapy also has the advantage of being firmly based in scientific methodology and has arisen out of basic, well-established psychological principles.

SUMMARY

The role of inhibition in psychotherapy is discussed. Five common criticisms of objective psychotherapy are examined and the conclusion reached that they are not damaging to either the theory or practice of non-analytic psychotherapy.

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SOUTH AFRICAN ORTHOPAEDIC ASSOCIATION

The Annual Congress of the South African Orthopaedic Association was held in Durban on 16-21 September 1957 in the course of and in association with the South African Medical Congress. The chair was taken by Mr. C. T. Moller, and 30 members were present, in addition to Sir Harry Platt (Manchester) and Mr. W. Gissane (Birmingham) as visitors.

Appointments

At the annual general meeting, which was held on 16 September, Sir Harry Platt and Mr. F. W. Fouché were elected Honorary Members of the S.A. Orthopaedic Association. The following were elected as office-bearers for the ensuing 2 years: *President*—A. J. Helfet (Cape Town); *Hon. Treasurer*—S. Sacks (Johannesburg); *Hon. Secretary*—G. Dommisse (Pretoria); *Committee members*—C. T. Moller (retiring President), J. M. Edelstein (Johannesburg), G. T. du Toit (Johannesburg), and J. G. du Toit (Pretoria). Prof. C. Lewer Allen (Cape Town) was elected to the editorial board for the ensuing 4 years.

Scientific Meetings

At the Plenary Session on 'The Surgery of Repair' held on 19 September, Mr. W. Gissane spoke on 'The Surgery of Injuries of the Hand'* and Sir H. Platt summed up the discussion.

At a combined meeting of the sections of Orthopaedics and Physical Medicine on 18 September Dr. J. Cyriax (London) spoke on 'Diagnosis at the Shoulder'.*

At a symposium on 'The Treatment of Pain' held on 18 September by the section of Neurology, Psychiatry and Neuro-surgery, Mr. A. Lewer Allen spoke on 'Pain in the Unstable Fracture'.

Clinical Meeting

On 17 September a clinical programme was presented at the Addington Hospital by the Durban members of the Orthopaedic Association:

Mr. R. Hill (*Durban*) showed a baby with congenital pes calcaneus plus a strange congenital undifferentiated band of tissue stretching from the tendo Achillis to the ischium in one continuous piece.

Mr. C. Kaplan (*Durban*) showed a female aged 18 years who had been unsuccessfully treated for a congenital dislocation of her left hip by two shelf operations at the age of 2½ years and at 4½ years. The discussion ranged around the treatment. She had no pain but did not like wearing a raised shoe to compensate for the 3 inches of shortening of the left leg. Some members suggested shortening the opposite leg for cosmetic purposes only. Others advised leaving her alone. Sir H. Platt advised a bifurcation osteotomy in order to postpone the onset of pain.

Mr. F. Hedden (*Durban*) showed an excellent result in a case in which the hamstrings had been transplanted into the patella for polio. A supra-condylar osteotomy had previously been done on the knee to correct a flexion contracture deformity of the knee.

He also showed a case of ununited fracture of the carpal scaphoid and the various methods of treatment for this condition were discussed by the meeting. Some members advised further immobilization in plaster. Others stressed the importance of reducing a fracture of the scaphoid by pulling on the thumb. Mr. Gissane advised immobilization of wrist and elbow to prevent rotation strains. After 6 weeks the plaster can be discarded. He stressed that it is bad teaching to recommend immobilization of a carpal scaphoid until it is radiologically united. Many people do normal work without the knowledge that they have ununited carpal scaphoids.

Mr. Helfet mentioned an operation to fuse the capitate to the scaphoid in cases of non-union.

Mr. G. Bickerton (*Durban*) showed a boy of 15 years who had presented with signs resembling tuberculosis of the hip with discharging sinuses and flexion contracture. He eventually proved to be a case of chronic appendix abscess and was cured by appendicectomy. No evidence of tuberculosis or actinomycosis was present.

Mr. J. Raftery (*Durban*) showed a case of pseudo-arthrosis of tibia in a male aged 11 years in whom he had obtained successful union with a massive cortical intramedullary bone graft supplied by the bone bank.

Mr. R. Hill (*Durban*) showed a case with traumatic paraplegia and calcification around the hip joint. The cause of calcification in paraplegics was discussed. Thrombosis of veins, trauma, and excessive physiotherapy, were mentioned by Mr. L. Mirkin as possible causes. Mr. Sacks mentioned cases he had seen and concluded that the aetiology was obscure and none of these theories of causation could be accepted. Mr. Helfet mentioned that in Holland it was held that sodium citrate taken by mouth would prevent this calcification.

SUMMARIES OF PAPERS

The following are summaries of some of the papers read at the meeting:

* To be published in this *Journal*.

'Giant-Cell Tumours' (Sir Harry Platt): section of Orthopaedics, 17 September

This neoplasm is comparatively rare and should be distinguished from other cysts of bone. One of these tumours occurs each year in each large teaching hospital. The speaker had only had 40 in 40 years. In the 19th century Paget, Nelaton and others regarded these tumours as essentially benign. Bloodgood had introduced the term 'Benign Giant-Cell Tumour'; Geschichter and Copeland (1930) reinforced the idea that it was fundamentally a benign tumour. Jaffe and Lichtenstein confirmed the view that it had an epiphyseal and not a metaphyseal origin.

There are 3 types, viz. benign, progressive and malignant. It occurs equally in both sexes and mostly in the long bones.

In the differential diagnosis, dystrophic cysts, atypical cysts and aneurysmal bone cysts should be distinguished. As regards prognosis, if giant-cell cysts are perfectly curetted, lasting cure can be obtained. Curettage on several occasions may lead to malignancy. If irradiated they may become malignant.

In 27 cases curetted and cauterized without bone grafting, 14 had given a lasting cure, 7 subsequently required amputations and 2 knee joints required fusion; 12 cases required excision and bone-graft replacement, e.g. at shoulder and wrist.

As regards treatment by irradiation, reports were conflicting. The speaker advocated primary surgery for accessible tumours. With irradiation much time is involved; the adjacent joint may be damaged; and there is the remote possibility of malignant change.

In the discussion Mr. Edelstein mentioned a case he had seen with giant-cell tumours in multiple bones. Sir Harry Platt said he could not recall having seen such a case. On the question of filling the curetted cavity with bone chips he stated that he did not do it unless the cavity was large and collapse of the cavity was imminent.

'Slipping or Displacement of the Vertebral End-Plates Simulating Disc Prolapse' (Mr. A. J. Helfet, Cape Town): section of Orthopaedics, 18 September

The story of two cases of young healthy lads aged 15 years and 16 years was described. They had developed acute pain in the back with radiation down both legs after a sudden strain at tennis in one case, and after lifting a heavy bag of wheat in the other.

They had flat, rigid spines with weakness of some of the leg muscles and sensory disturbance in the legs. Conservative treatment was of no avail. At operation the first case revealed a sequestrated intervertebral disc at the L4-L5 level attached to a ledge of cartilage which appeared to be attached to a posteriorly displaced vertebral end-plate. The disc was removed and the projecting end-plate was nibbled away with bone-nibbles. The patient made a complete recovery and now takes part in rugby and high-diving.

The second case had a slight pyrexia and could hardly move about in bed. At operation a bulge was found at the 4th lumbar space with a posteriorly projecting ridge above it. An attempt was made to grasp this projection but it slipped forward between the vertebrae and the bulging disc disappeared with it. No attempt was made to remove the disc or slipped end-plate; they appeared to have slipped back into place. The patient was kept in bed for 14 days after operation and, despite a mild pyrexia for 5 days, all the motor and sensory disturbances disappeared. An X-ray of the spine taken 5 months later showed a narrowed intervertebral space with some marginal sclerosis and possibly some slight erosion. It was thought that these appearances probably indicated some mild infection of that region.

Mr. Helfet showed the X-rays of these cases and by means of line-drawings depicted the normal development of the vertebral end-plates and cartilaginous rings.

In the discussion Mr. Sacks mentioned that primary intervertebral-disc infection sometimes occurs in children and that the oedema and vascularity of the infection might possibly lead to the pathology described by Mr. Helfet.

Mr. Edelstein showed some X-rays of cases with anterior subchrondral protrusion of discs and wondered if these posterior protrusions described by Mr. Helfet could not be described in the same light.

A Study on Congenital Displacement of the Hip (Mr. D. J. Retief, Johannesburg): section of Orthopaedics, 18 September

By means of X-rays and diagrams emphasis was placed on the necessity for open reduction and removing the intervening limbus if closed reduction fails.

A case was also made for arthrography of the hip joint, and some very good X-ray slides were shown. It was mentioned that at some clinics in England limbectomy was almost a routine procedure.

In the discussion Mr. Edelstein stated that before he could accept the fact that the limbus presented an insuperable difficulty to closed reduction, he would like to know how the limbus was formed. Is the mass of fibrous tissue or invaginated capsule the limbus?

Mr. Möller said that when he operated and removed the obstructing material he usually found only a frail piece of tissue. Even when this was removed the head of the femur still did not seem to settle deeply into the acetabulum—unless the femur was placed in gross internal rotation. He found that only 30% of cases could not be reduced satisfactorily by closed reduction.

Sir H. Platt stated that there were a great many different types of congenital dislocations of the hip. It is not true that a complete dislocation first goes through a stage of subluxation. Subluxation is a type of its own. Arthrography is of no use and is not a great need. He had done 140 open reductions and less than 30% of his cases had required this operation. In children under 3 years of age closed manipulation produces a good functional and anatomical result. Derotation osteotomy is very rarely necessary in young infants. It is only done at a very much later stage. The question of which hip to treat primarily by open reduction is still unanswered.

The Treatment of Poliomyelitis in the Early Stages (Mr. C. J. Kaplan, Durban): section of Orthopaedics 20 September

The complications occurring in the early stages of poliomyelitis were mentioned. In bulbar polio there was a group of cases with upper cranial nerve paralysis, and a group with lower cranial nerve paralysis, including laryngeal and deglutition paralysis. The respiratory-centre group, the circulatory group and the encephalitic group were also mentioned.

In the high cervical group of paralysis the respiratory muscles were involved.

The treatment of these various complications was discussed.

Slides were shown of the various respiratory instruments and aids employed. Mention was made of the method used to teach patients to perform glosso-pharyngeal breathing to enable them to leave their respirators.

Pictures were shown of the cervical braces or 'halos' used in cases where the spine had been fused to prevent the head from falling forwards.

Orthopaedic surgeons should have direct access to polio cases from the commencement of the illness.

PASSING EVENTS : IN DIE VERBYGAAN

Lede word daaraan herinner dat hulle die Sekretaris van die Mediese Vereniging van Suid-Afrika, Posbus 643, Kaapstad, sowel as die Registrateur van die Suid-Afrikaanse Mediese en Tandheelkundige Raad, Posbus 205, Pretoria, moet verwittig van enige adresverandering.

Versuim hiervan beteken dat die *Tydskrif* nie afgelewer kan word nie. Dit het betrekking op lede wat oorsee gaan sowel as dié wat binne die Unie van adres verander.

REVIEWS OF BOOKS : BOEKRESENSIES

THE PERSON BEHIND THE DISEASE

The Person Behind the Disease. By Julius Bauer, M.D., F.A.C.P. Pp. vii + 136. $3.50. New York and London: Grune & Stratton, Inc. 1956.

Contents: Preface. I. Etiologic and Pathogenetic Factors in the Diseased Person. II. Uniqueness of the Individual. III. Pathogenic and Nonpathogenic Mutations. IV. Clinical Syndromes and Irregular Polygenopathies. V. Constitutional Biologic Organ Inferiority. VI. Holistic Medicine: The Parts and the Whole. VII. Psychologic Medicine.

Dr. Bauer sets out, as he says in the preface, 'to elaborate the fundamental principles of a holistic concept of medicine'. First, there are extrinsic causes of disease. These have, up to recent times, received most attention and study. Even in this field many questions remain unanswered. Why, when one person gets infected with the treponema pallidum, does he get tabes whilst another suffers from bone syphilis? And why, after tonsillectomy, when more than a third of the subjects have a temporary bacteraemia do some cases develop subacute bacterial endocarditis whilst most escape this complication. He discusses genetic constitution not only as regards gross physical development but also as it shows itself in enzymic defects. What is the basis of heredo-familial degenerations and what is there in Gower's concept of abiotrophy! Dr. Bauer enumerates a number of syndromes in many of which several, apparently unrelated abnormalities occur, e.g. the Laurence-Moon-Biedl, the Turner's and the Peutz-Jegher syndromes. Not least important is the psychological side of the person.

To many of the questions asked there simply are no answers. In so short a book many aspects are no more than touched on. There is every now and then an abrupt jump from one point to another: the theme is not smoothly developed. Surprisingly, allergy is dealt with only in the preface.

F.F.

TRIALS OF EVANS AND CHRISTIE

Trials of Evans and Christie. Edited by F. Tennyson Jesse. Pp. c + 379. 16 Illustrations. 30s. London, Edinburgh, Glasgow: William Hodge & Co. Ltd. 1957.

Contents: Introduction. Leading Dates. *Trial of Evans:* Arraignment. Legal Argument. Opening Speech for the Prosecution. Evidence for the Prosecution. Evidence for the Defence. Closing Speech for the Prosecution. Speech for the Defence. Charge to the Jury. Verdict. Sentence. *Trial of Christie:* Arraignment. Opening Speech for the Prosecution. Evidence for the Prosecution. Opening Speech for the Defence. Evidence for the Defence. Evidence in Rebuttal for the Prosecution. Closing Speech for the Defence. Closing Speech for the Prosecution. Charge to the Jury. Verdict. Sentence. Appendix I—Appeal by Timothy John Evans. Appendix II—The Scott Henderson Report. Appendix III—Debate in the House of Commons, 29th July, 1953. Appendix IV—Supplementary Report by Mr. Scott Henderson. Appendix V—Debate in the House of Commons, 5th November, 1953.

The Trial of John Reginald Halliday Christie at the Old Bailey shook the public. Not only because the man on trial was the self-confessed murderer of at least six people (and possibly more) but also because there emerged at trial the deadening possibility that an innocent man Timothy John Evans had been sentenced to death and executed in 1950, for a murder that was possibly done by Christie.

The facts are well known. Both Christie and Evans lived together in the same apartment house where Evans' wife was found strangled, together with the body of his 14 month old daughter who had also been strangled. Strangulation was the method used by Christie in all his murders. An extraordinary coincidence if it were to be that two murderers were living in die same premises, both using strangulation as a means of killing their victims.

The investigation resulting in Evans being brought to trial was initiated by a confession made by him to the police. At his trial Christie was an important crown witness against Evans. In one statement (among several) made to the police Evans had implicated Christie. What was not known at the time was that in the garden of the same house, were buried the skeletons of two women murdered by Christie by strangulation in 1943 and 1944.

The medico-legal interest in the trial of Christie was his mental and psychological state. It was established by the psychiatric evidence that he suffered from hysteria and that he was a necrophilist. His murders had a marked sexual element. Intercourse occurred while his victims were dying or dead. Christie was duly convicted and hanged.

The defence taken was that of insanity. In English law such defence will only succeed if brought within the limits of the well known M'Naghten rules. The accused must either 'not know the nature and quality of the act at the time he performed it or not know that it was wrong'. The psychiatric evidence could not satisfy such requirement in respect of Christie's mental state. South African law is wider than English law and a defence of irresistible impulse based on mental disease, if such impulse prevents a person from controlling his conduct, may be taken. One may speculate whether Christie's sexual perversion taken together with his hysterical state would have brought him within the ambit of a defence in a South African court. This is an interesting problem for the psychiatrist.

The trial of Christie led to a debate in the House of Commons, and an inquiry into the deaths of Evans' wife and child, which was also debated. The report is given in full as are also the debates as reported in Hansard. The complete records of both trials are included in the book and a first class analysis in the form of a long introduction by the authoress, Mrs. F. Tennyson Jesse.

This book, though it contains largely verbatim reports and speeches, is better than any thriller. Medically it provides a fascinating exercise in psychiatric diagnosis. It proves once again that no doctor can be too careful when called upon to give evidence. A man's life can hang on a medical thread.

A.P.

THE MEDICAL ANNUAL

The Medical Annual. Seventy-fifth issue. Edited by Sir Henry Tidy, K.B.E., M.A., M.D. (Oxon.), F.R.C.P. and R. Milnes Walker, M.S. (Lond.), F.R.C.S. Pp. xl + 570. Plates LII. Bristol: John Wright & Sons Ltd. 1957.

Contents: Contributors and their Contributions. List of Plates. Publisher's Note. Introduction by the Editors. Review of the Year's Work. *Special Articles:* The Modern Theory of Blood Coagulation. Hypothermia. Nutrition and Vitamins. Prostatic Enlargement. The Practitioners' Index: Recent Pharmaceutical and Dietetic Preparations, Medical and Surgical Appliances, etc. Books of the Year. General Index.

The Medical Annual, under the joint editorship of a leading British physician and a leading British surgeon, continues in its well-known way for the 75th time. This annual review attempts to encompass a year's advances in the whole field of the art and science of medicine. It is rather difficult to know to whom to recommend it. Is it for the general practitioner? He will find plenty to interest him, but seldom enough of any one subject to help him. He will find many rarities which he will probably ignore. The specialist will find some useful references in certain fields, but will be disappointed at the amount which remains, rather arbitrarily, unmentioned. The medical student will value the four special articles, which are on the subject of blood clotting, hypothermia, nutrition, and prostatic enlargements, but he will certainly not buy the book. It seems to this reviewer that the most value is obtained from the sort of article like that by Meadows on myasthenia gravis, which occupies 6 pages and summarises thoroughly the important modern views on this disease, quoting only a small number of references. For more complete coverage of the literature of each speciality, the seeker for knowledge could then turn to annuals dealing only with his special field.

It is a good hardy annual, and will claim its usual faithful followers. It has, however, reached the age at which, if the mixed metaphor be excused, a face-lift might be desirable.

W.J.

BEDSIDE DIAGNOSIS

Bedside Diagnosis. Fourth Edition. By Charles Seward, M.D., F.R.C.P. (Edin.). Foreword by Lord Cohen of Birkenhead, M.D., D.Sc., LL.D., F.R.C.P., F.A.C.P., F.F.R. Pp. xxiv + 430. 21s. net + 10d. Postage Abroad. Edinburgh and London: E. & S. Livingstone Ltd. 1957.

Contents: Introduction. I. Psychogenic Symptoms. II. Some General Considerations regarding Pain. III. Head Pain. IV. Thoracic Pain. V. Epigastric Pain

VI. Umbilical Pain. VII. Hypogastric Pain. VIII. Lateral Abdominal Pain. IX. Anaemia. X. Epistaxis. XI. Haematemesis. XII. Haematuria. XIII. Haemoptysis. XIV. Haemorrhagic Disease. XV. Cough. XVI. Dyspnoea. XVII. Tachycardia. XVIII. Dysphagia. XIX. Vomiting. XX. Diarrhoea. XXI. Jaundice. XXII. Debility and Loss of Weight. XXIII. Pyrexia. XXIV. Normal Values. Index.

Since this book first appeared in 1949 (and was reviewed in the *Journal*), it has gone through 4 editions and has been translated into Spanish and Portuguese. Such a demand indicates that, with this book, Dr. Seward has indeed filled a need which he felt existed both for the medical student and for the doctor.

The layout of the book is unchanged in that the main presenting symptoms of disease of the different bodily systems are presented at the beginning of each chapter and are dealt with on a standardized plan beginning with a synopsis of the causes of the symptom, the physiology of the symptom and then the diagnostic approach. The diseaes themselves are but briefly described, and obviously the book must be used in conjunction with larger standard text books of Medicine.

With each successive edition small additions to the subject matter has been made and the book remains thoroughly up to date without any appreciable increase in bulk. A very useful addition, at the end, is a chapter on Normal Values.

The main faults of the book are the inevitable results of compression, so that no adequate evaluation of the importance of various conditions in the causation of different symptoms is possible. For example, in discussing headaches, one is left with the impression that any elevation of blood pressure above the arbitarily chosen normal of 150/100 may be responsible for headaches. The description of some diseases also is so brief that the conditions are scarcely recognizable. However, there is a commendable emphasis on the importance of psychogenic symptoms, and the opening chapter on Psychogenic Symptoms is particularly well presented.

There can be no doubt that Dr. Seward's book will continue to maintain and even increase its popularity. One can recommend it especially to the student in his first clinical years.

H.M.

PARTICULATE CLOUDS

Particulate Clouds: Dusts, Smokes and Mists. By H. L. Green M.A. (Cantab.), F. Inst. P. and W. R. Lane, B.Sc. (Birm.), F. Inst. P. Pp. xix + 425. Illustrated. 70s. net. London: E. & F. N. Spon, Ltd. 1957.

Contents: Foreword. Preface. Acknowledgements for Figures and Tables. Literature Abbreviations. *Section 1.* Introduction. *Section 2.* Production of Particulate Clouds. *Section 3.* Some Physical Characteristics. *Section 4.* Optical Properties. *Section 5.* Coagulation. *Section 6.* Deposition and Filtration. *Section 7.* Sampling and Estimation. *Section 8.* Diffusion in the Atmosphere. *Section 9.* Collection. *Section 10.* Health Hazards. *Section 11.* Atmospheric Pollution. *Section 12.* Aerosols in Nature. *Section 13.* Uses of Particulate Clouds. Author Index. Subject Index. Plates.

Pollens and some other allergens spread as air-borne particles; tobacco smoke is carried to the lungs and to the audience in theatres in the same kind of way. Bacteria may be spread as particulate clouds. Other particulate clouds, under the name of 'smog', have in recent years become notorious health hazards. Examples of importance in public health are the spread of radio-active isotopes in the atmosphere by nuclear explosions and the attempts to reduce the incidence of silicosis in South African gold mines. In part I of this book the authors deal with the physics and the physical chemistry of clouds of small particles or droplets. Production of clouds artificially and in nature, their optical properties, the rates of coagulation of the particles, deposition and filtration and methods of sampling are all treated, as also diffusion processes in the atmosphere. Much of the treatment is quantitative and is not easily understood without a considerable knowledge of physics and mathematics. Results of calculations are, however, often presented in a simple way by means of graphs or tables. This part of the book—more than half of the whole volume—would be valuable to public health officers and to medical research workers mainly as a work of reference. As such it is authoritative and covers a very wide field, including much not likely to be of direct interest to doctors.

Part II is, in the main, easily read. It gives very useful descriptions of methods of collecting particles from clouds and there is a chapter on health hazards, including tables of properties of a number of radioactive aerosols. Such topics as the respiratory retention of bacterial aerosols, tobacco smokes, and fire and explosion hazards are also treated. Atmospheric pollution, smog and chemical contaminants are treated in some detail. Then, there are descriptions of therapeutic applications, such as inhalers and atomizers.

On the whole this is a most valuable book with a very useful bibliography. It is an essential for the library of a medical school and for research workers in certain fields of medicine. Some chapters should be of interest to all medical men. It is well printed and the plates and diagrams are very well reproduced. W.S.

ATLAS OF MUSCLE PATHOLOGY

An Atlas of Muscle Pathology in Neuromuscular Diseases. By J. Godwin Greenfield, M.D., G. Milton Shy, M.D., Ellsworth C. Alvord, Jr., M.D. and Leonard Berg, M.D. Photomicrographs by Fred H. Meiller. Pp. ix + 104. Illustrations, some in colour. 45s. net + 10d. Postage Abroad. Edinburgh and London: E. & S. Livingstone Ltd. 1957.

Contents: Part I. Histopathological Reactions of Muscle. 1. Structural Changes in the Muscle Fibre: Loss of Cross-striation, Cloudy and Granular Changes, Floccular Changes and Phagocytosis. 2. Changes in Muscle Nuclei. 3. Changes in Fibre Size. 4. Basophilic Fibres with Vesicular Nuclei and Prominent Nucleoli. 5. Ringed Fibres. 6. Sarcoplasmic Masses. 7. Changes in Interstitial Tissues: Collagen and Fat. 8. Leucocytic Infiltrations. 9. Changes in Muscle Spindles and Peripheral Muscular Nerves. 10. Pattern of Lesion. *Part II. Clinico-Pathological Correlations.* 11. Clinical Classification of Neuromuscular Diseases. 12. Distal Muscular Syndromes. Large Groups of Small Fibres and Abnormal Peripheral Muscular Nerves and Muscle Spindles. 13. Myotonic Syndromes. Ringes Fibres, Sarcoplasmic Masses and Internal Nuclei. 14. Proximal Muscular Syndromes. Abnormally Large Fibres, Structural Changes, Phagocytosis, Basophilic Fibres with Vesicular Nuclei and Prominent Nucleoli, Leucocytic Infiltrations and Abnormal Collagen and Fat. 15. Myasthenia Gravis: Lymphorrhages and other Abnormalities. Summary. Conclusions. Appendix I: Methods and Techniques. Appendix II: Cross-index of Cases. Bibliography and References. Index.

This work is the result of a study by four independent investigators who set out to determine how much the pathologist, with ordinary techniques, could contribute to the differential diagnosis of neuromuscular diseases. Muscle biopsies from 121 cases of neuromuscular disease were examined, at first by each investigator individually and later by the group, in an attempt to define the fundamental histological changes that occur in diseased muscles and to correlate these findings with clinical observations.

A pathologist's interpretation of a histological section must, of necessity, be influenced by the clinical information made available to him. In a field where much ignorance and confusion still exists as to the significance of histological appearances there is, however, a great deal to be said for a 'blind' study of the histological sections by the pathologist lest, not only his interpretation, but even his description be influenced by the clinical diagnosis. Awareness of this has led the authors to divide their atlas into two parts.

In Part I the various types of histopathological changes that occur in muscles are defined and illustrated without reference to clinical data. The illustrations, many of which are in colour, are of a high standard. One is particularly impressed by the author's deliberate use of simple descriptive histological terms, and the avoidance of terms (such as atrophy or degeneration) which carry certain pathogenetic implications that may not always be correct.

In Part II the authors define in simple terms the various clinical types of neuromuscular disease and attempt to correlate the histological changes previously described with the clinical findings. No single change was found to be specific for any disease. Certain combinations of changes were suggestive (such as the presence of ringed fibres, sarcoplasmic masses, large muscle fibres and many internal muscle nuclei in dystrophia myotonica) but the authors are the first to admit the limitations of muscle biopsy in the differential diagnosis of many of the neuromuscular disorders. One feels, with the authors, that further study of more cases will solve some of the problems in this field. The authors' description and evaluation of the histological changes seen in muscle biopsies will serve as a valuable basis for such studies and as a welcome guide to the pathologist engaged in routine diagnostic pathology.

An appendix describes the technique of muscle biopsy, and pathologists the world over will appreciate the advice given to the operator on how to avoid the distortion which may be produced by the injection of local anaesthetic into the muscle or by crushing or tugging.

The mode of presentation of the subject matter in this book is perhaps rather unorthodox, but the final product is one which should be welcomed, particularly by pathologists, but also by all who are interested in neuromuscular disorders. M.S.